



An investigation into a self-regulated approach to repeated sprint
exercise in elite youth association football players

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Abstract

Recent studies have applied an individualised approach to repeated sprint exercise (RSE), whereby participants are given the freedom to self-determine inter-interval recovery periods based on individual perceptions of recovery (Glaister *et al*, 2010; Phillips *et al*, 2014). These studies have suggested that such an approach may be a useful alternative to RSE with the aim of maintaining sprint performance. However, these studies have been conducted exclusively on adults, with no research conducted in young athletes. The aim of the present thesis was to evaluate the influence of using self-selected recovery periods with the aim of maintaining sprint time on performance and the physiological responses to repeated sprint exercise (RSE) in elite youth association football players. A second aim of the thesis was to assess the influence of maturation on the ability to self-determine recovery intervals between sprints. Chapter three assessed performance during a 10 x 30 m sprint task with a 30-second standardised recovery (SR) period, using self-selected recovery with no external cue (NEC), and facilitated by the use of a perceived readiness (PR) scale to assist with self-guidance of recovery intervals, whilst also examining differences in performance and recovery duration between more and less mature players. Twenty-eight elite youth association football players (aged 13 ± 0.9 years) were recruited for the study, with participants split into a more mature (aged 14 ± 0.5 years) and less mature group (aged 13 ± 0.9 years) based on stage of growth in relation to peak-height-velocity. The results demonstrated that participants displayed a tendency to underestimate the recovery time required to maintain performance during the NEC and PR trials, resulting in impaired performance when compared with the SR trial, in which recovery intervals were significantly longer and performance was better maintained. Despite exhibiting less fatigue when SR periods were used, less mature participants displayed an impaired ability to guide recovery intervals with NEC, as displayed by the moderately higher sprint decrement compared with the more mature group. Chapter four evaluated the physiological, neuromuscular and perceptual responses to RSE with self-guided (SG) recovery periods compared with a 30-second standardised recovery (SR) period in 11 participants (aged 13 ± 0.9 years). This study suggests that using SG recovery induces a significantly higher physiological and metabolic stress on participants, with significantly higher post-exercise blood lactate values and moderately higher average HR values compared with the SR trial, likely due to the significantly shorter recovery periods used. These studies provide novel insight into the influence of using self-selected recovery periods on performance and the physiological response to RSE in young athletes.

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Abbreviations

- CO₂ - Carbon dioxide
- CNS - Central nervous system
- HR - Heart rate
- NEC - No external cue
- O₂ - Oxygen
- PCr - Phosphocreatine
- PHV - Peak height velocity
- PR - Perceived readiness
- RPE - Rate of perceived exertion
- RPE - Repeated sprint exercise
- SG - Self-guided
- SR - Standardised recovery
- VO_{2max} - maximal oxygen uptake

Chapter 1 - Introduction

In team sports characterised by repeated high-intensity efforts, such as association football, field hockey and rugby, repeated sprint training is commonly implemented amongst practitioners as a means of improving physical performance. Characterised by repeated short sprints (3 – 7 s in duration) separated by brief recovery periods (≤ 60 s), repeated sprint training has been shown to induce numerous physiological and biomechanical adaptations deemed specific to team sports, such as improvements in $\text{VO}_{2\text{max}}$, maximal sprint speed and jump height (Taylor *et al*, 2015). The traditional approach to repeated sprint training in research and in practice is to separate inter-interval recovery periods with work-to-rest ratios, or with a standardised recovery period. However, given that recovery during repeated sprint exercise is an aerobic process, it follows that individual differences in oxidative capacity will influence the ability to recover between sprints in order to limit fatigue (Glaister *et al*, 2010). In order to optimise the physiological stimulus from repeated sprint training, providing individualised recovery periods may prevent players from receiving insufficient or excessive recovery periods between sprints, which may in turn compromise desired training outcomes (Edwards *et al*, 2011). However, this approach may be particularly difficult logistically when training with groups of athletes, as is often the case in team sports.

In light of this, recent studies have implemented a self-regulated approach to repeated sprint training, whereby participants are given the freedom to determine their own recovery periods based on their perceived readiness to recommence sprint exercise. This approach requires individuals to accurately perceive physiological feedback in order to gauge their level of recovery in the context of the task. Studies implementing this recovery modality during repeated sprint exercise have suggested that this may be a useful training tool, with participants displaying the ability to accurately and consistently govern recovery periods in order to maintain performance during successive sprints (Glaister *et al*, 2010; Phillips *et al*, 2014). Furthermore, it has been suggested that using this psychophysiological approach to repeated sprint exercise may facilitate greater self-awareness amongst athletes through an improved understanding of physical capabilities (Edwards *et al*, 2011). However, this research has been conducted exclusively on adult populations, with no studies assessing self-regulated repeated sprint exercise in young athletes.

During each self-guided inter-interval recovery period, the length of recovery duration is likely to be influenced by afferent feedback from a range of physiological variables, such as blood/muscle pH, cardiorespiratory sensations and core temperature, which influence the conscious perceptions of exertion and perceived readiness for recommencing sprint exercise (Edwards *et al*, 2011). Furthermore, as well as involving sensory-perceptual elements, the ability to accurately govern recovery periods between sprints is likely to be influenced by cognitive factors, such as planning, anticipation, logical reasoning, and the ability to form internal spatial representations of a given task (Micklewright *et al*, 2011). In young athletes, stage of maturation has been shown to influence the physiological responses to repeated sprint exercise, with less mature children commonly displaying an enhanced ability to recover between bouts of sprinting compared with more mature adolescents, likely due in part to their higher affinity for aerobic metabolism and lower reliance on anaerobic glycolysis (Ratel *et al*, 2006). Thus, from a physiological perspective, stage of maturation may influence the amount of recovery time taken during self-regulated repeated sprint exercise. From a cognitive perspective, however, differences in the level of intellectual development and/or experience may also influence the ability to accurately perceive readiness for recommencement of sprint exercise in order to maintain performance during successive sprints (Micklewright *et al*, 2011). Information on this area may have practical implications through allowing practitioners to make more informed decision making when considering implementing this form of repeated sprint exercise amongst specific age groups, whilst also giving insight into the influence of maturation on the regulation of intensity and recovery during self-regulated repeated sprint exercise.

Furthermore, integral to the implementation of training modalities aimed at inducing specific adaptations is an understanding of the acute physiological, metabolic and neuromuscular responses evoked. However, limited research on these responses to self-regulated repeated sprint exercise currently exist, with physiological data in previous studies limited to HR (Glaister *et al*, 2011; Phillips *et al*, 2014). An appreciation of the influence of using self-guided recovery periods on performance during repeated sprint exercise, alongside an assessment of the physiological responses to this training modality, may have important practical implications through providing a more comprehensive understanding of the applicability of self-regulated repeated sprint exercise amongst young team sport athletes with the aim of producing specific adaptations.

The aim of this thesis was to evaluate performance, physiological responses, and the influence of maturation on the ability to self-govern recovery intervals during self-regulated repeated sprint exercise with the aim of maintain sprint performance in elite young association football players. Performance and recovery variables were measured in young athletes of different stages of maturation in Chapter 3, while the physiological, neuromuscular and perceptual responses to self-regulated repeated sprint exercise were assessed in Chapter 4.

Chapter 2 – Literature Review

2.1 - Introduction

In recent years, there has been an increase in the number of research studies investigating the role of the central nervous system (CNS) in the development of fatigue. Early studies suggested that fatigue, defined as the failure to maintain the required or expected power output (Edwards, 1983), was a result of physiological impairment, in which mechanisms originating in the periphery are responsible for inhibitions in forceful muscle contractions, thereby reducing physical work capacity and performance (Waldron & Highton, 2014). Peripheral factors thought to induce fatigue include substrate depletion including glycogen and creatine phosphate (Krustrup *et al*, 2006), metabolite accumulation, such as lactic acid and concomitant reductions in blood and muscle pH (Fitts, 1994), disturbances in ion homeostasis, and impaired excitation of the sarcolemma (Mohr *et al*, 2005).

The peripheral model of fatigue has also been described as the “catastrophe” theory (Noakes, 2004), due to the notion that forceful muscular contractions are inhibited and ultimately terminated as a result of one or more system failures, in which the physiological and biochemical limits of the body are exceeded, resulting in an involuntary termination of exercise (Edwards, 1983). While evidence has displayed the attenuating effects of peripheral fatigue on performance (e.g. Taylor *et al*, 1997; Hunter *et al*, 2003), contemporary research suggests that the mechanisms which induce fatigue are more complicated, and involve a complex interplay between both peripheral and central factors (Amann, 2011).

Recent evidence suggests that rather than peripheral factors directly causing fatigue, information regarding exercise-induced biochemical changes within the muscle are relayed to the CNS through metabosensitive thin-fibre muscle afferents (St. Claire Gibson & Noakes, 2004). This afferent physiological feedback results in adjustments in the efferent, feedforward control of motor unit recruitment from the motor cortex of the brain, termed central motor drive, causing alterations in the type, rate and frequency of motor unit recruitment (Thompson *et al*, 2014) as well as the subjective perception of the demands of a given task (Tucker, 2008). The goal of this feedback-feedforward control loop is to prevent the level of peripheral fatigue from exceeding an individual “critical threshold”, and causing the cessation of exercise (Amann, 2011). Furthermore, this

central control of exercise intensity serves to ensure that homeostasis is maintained within tolerable limits, responding to feedback from various receptors such as chemoreceptors, mechanoreceptors and thermoreceptors to protect the body from potentially harmful damage through over-exertion (Micklewright *et al*, 2009).

These advancements in our understanding of the role of the CNS in the development of fatigue has led to a shift in focus from the physiological aspects which *limit* exercise performance, to those aspects that *regulate* exercise performance. In turn, this has prompted research investigating pacing strategies in laboratory (e.g. Turnes *et al*, 2014) and competitive settings, including both individual (e.g. Saavedra *et al*, 2012) and team sports (e.g. Aughey *et al*, 2013). The term pacing strategy refers to the distribution of work or pattern of energy expenditure during exercise (Abbiss & Laursen, 2008). During self-paced exercise in which individuals are free to set their own pace, exercise intensity is regulated in anticipation of the demands and expected duration of a task, and on continual physiological feedback from the periphery (Tucker, 2009).

Several internal and external factors are thought to influence pacing strategies, such as environmental conditions, including temperature (Duffield *et al*, 2009) and altitude (Clark *et al*, 2007), psychological factors such as motivation and arousal (Tucker & Noakes, 2009), presence of competitors (Lambrick *et al*, 2013) and knowledge of endpoint (Billaut *et al*, 2011) as well as mode, distance and duration of exercise (Billaut *et al*, 2011). One area which has received little attention in the literature is the influence of maturity.

Evidence has shown that stage of maturation influences the physiological and metabolic responses to exercise (e.g. Lazaar *et al*, 2002). A consistent finding throughout the literature is that younger, less mature adolescents (aged 11-13) are better able to resist fatigue during high-intensity exercise (Ratel *et al*, 2006), whilst also recovering quicker between bouts of high-intensity intermittent exercise compared with older, more mature adolescents (aged 14-17) (Lazaar *et al*, 2002). There are several explanations for the observed differences in fatigability between less and more mature adolescents. Namely, less mature children have lower muscle mass in comparison with more mature adolescents and thus generate lower levels of absolute power during high-intensity exercise, which has been shown to be related to reduced fatigability and faster recovery from high-intensity exercise (Ratel *et al*, 2004). Furthermore, maturational related differences in central and peripheral physiology are also likely to play an influential role in the differences in fatigability. For example, Buchheit *et al* (2011) found that pre peak-

height-velocity (PHV) adolescents displayed a faster heart rate recovery and parasympathetic reactivation following a graded running test to exhaustion.

Given that pacing of effort during exercise involves afferent feedback relating to physiological and metabolic parameters, the magnitude of which may be influenced by maturation, it could be argued that adolescents at different stages of maturity may display differences in self-regulation of intensity during exercise, particularly during high-intensity intermittent exercise, such as is found in team sports. In order to fully understand the potential influence of maturity on pacing strategies, the neurophysiological and psychological mechanisms involved in pacing must first be explored. Thus, the purpose of this review is to examine the mechanisms that influence the regulation of exercise intensity, before assessing how maturation may impact on these mechanisms. The cognitive and sensory-perceptual elements of pacing will be explored, as will the external factors which influence pacing strategies.

2.2 - Intrinsic Regulation of Pacing Strategy during Exercise

Peripheral fatigue has been defined as any biochemical change within the working muscle resulting in an attenuated response to neural excitation (Amann, 2011). The traditional peripheral model of fatigue proposed by A.V. Hill (1924) implies that fatigue originates in the periphery, i.e. the working muscles. The model predicts that exercise performance is limited by the accumulation of metabolic by-products, and the depletion of energy substrates, which results from the onset of anaerobiosis (Fitts, 1994). These by-products, such as lactic acid, were seen as “poisonous” to the muscles, causing alterations in contractile function and resulting in involuntary termination of exercise (St. Claire Gibson & Noakes, 2004).

Several other peripheral factors have been suggested to be responsible for impairments in performance during short duration high-intensity exercise (e.g. intermittent sprint activity), and prolonged endurance type exercise (e.g. a 20km run). One proposed mechanism of fatigue during high-intensity exercise is the accumulation of potassium (K^+) in the muscle interstitium. Interstitial K^+ accumulation is thought to be related to the exercise-induced reductions in intramuscular pH, causing K^+ loss from the muscles through K_{ATP} channels which open in response to a reduction in pH (Mohr *et al*, 2005). *In vitro* evidence has shown that levels of interstitial K^+ accumulation typically found during short-duration high-intensity exercise (Nielsen *et al*, 2004) bring about electrical

disturbances in the muscle cell and reductions in force generating capacity (Cairns & Dulhunty, 1995). Reductions in muscle creatine phosphate concentrations have also been cited as contributing to fatigue during high-intensity exercise (Greenhaff *et al*, 1994). Evidence to support this comes from the low levels of intramuscular creatine phosphate found at the point of fatigue following high-intensity exercise (Krustrup *et al*, 2006), and from improvements in performance during high-intensity intermittent exercise following a period of creatine supplementation (Greenhaff *et al*, 1994). During more prolonged exercise, depletion of intramuscular glycogen stores is thought to induce fatigue and impairments in performance. Support for this notion comes from research which has found improvements in performance following increases in muscle glycogen levels through carbohydrate loading (Wright *et al*, 1991).

Counter arguments to the assertion that peripheral factors are responsible for the development of fatigue have been cited and propose a greater involvement of the CNS (Noakes, 2005; Gandevia, 2001). It is widely accepted within all manner of sports and exercise modalities that participants will adopt a pacing strategy that allows them to successfully and safely complete the given task (Thompson *et al*, 2014). The traditional model of fatigue, however, appears limited in its ability to explain how, during endurance based exercise, athletes are able to increase their speed during the latter stages, despite the levels of “poisonous” metabolites being at their highest, and energetic substrate levels being at their lowest. Furthermore, the peripheral model predicts that at the time of exhaustion during any form of exercise, all available motor units must be recruited or have been exhausted during subsequent activity (Noakes *et al*, 2005). Research has shown however that this may not be the case. During prolonged exercise the percentage of available motor units recruited at the point of exhaustion was found to be around 30% in recreational athletes (St. Claire Gibson *et al*, 2001), and even during short-duration maximal isometric contractions, muscle is not fully recruited (Yue *et al*, 2000). These observations have led to the hypothesis that the degree of motor unit recruitment during exercise is mediated by a “central governor” in the brain, which directs exercise intensity through subconscious alterations in skeletal muscle fibre recruitment (Lambert *et al*, 2005). The purpose of this regulation of muscle recruitment is to prevent the premature onset of fatigue, and ensure that homeostatic control of central and peripheral physiological systems is maintained, thus preventing harmful damage to the body.

This hypothesis, known as the Central Governor theory, has been widely debated in the scientific literature (Shepard, 2009; Hopkins, 2009; Marcora, 2008). One of the main

arguments opposing the theory is that, despite the apparent dismissal of peripheral fatigue by those advocating the Central Governor theory (Noakes, 2011), a number of studies have shown that during short-duration maximal exercise power output declines despite the level of electrical activation to the muscle remaining unchanged (e.g. Hunter *et al*, 2003), suggesting that peripheral factors are responsible for the decrements in performance. Moreover, Presland *et al* (2005) found that peripheral fatigue accounted for two-thirds of the 30% reduction in maximal voluntary isometric force following prolonged cycling to exhaustion, with central fatigue accounting for one-third. Furthermore, while it has been suggested that the central governor serves to protect the body from catastrophic system failure and ensure the maintenance of homeostasis (Noakes, 2011), there are examples of athletes experiencing exercise-induced conditions such as hyperthermia or myocardial fibrosis (Hopkins, 2009). Therefore, if a central governor in the brain which serves to protect the body from harmful damage does exist, it does not appear to be infallible. This is because an infallible central governor would sense the threat to homeostasis and reduce intensity accordingly before harmful damage occurred.

While the debate on the Central Governor hypothesis continues, there appears to be a general agreement within the scientific community that the brain and CNS play an important role in regulating exercise intensity and pacing (Cairns, 2011), and that pacing is regulated in an anticipatory manner, both to optimise performance and to prevent large homeostatic perturbations (de Koning *et al*, 2011). Furthermore, there is evidence that afferent sensory feedback from various physiological systems play an important role in the brains regulation of motor unit recruitment (Amann, 2011).

Afferent Feedback

During exercise, the CNS integrates signals sent from the periphery regarding the physiological state of the body. These afferent signals are derived from receptors such as mechanoreceptors and chemoreceptors in the body. Rather than metabolic intermediates directly causing fatigue in the periphery, these metabolites are suggested to have an important role as signalling agents to the brain (Roelands *et al*, 2013). Afferent signals relating to ventilation, temperature, cerebral and arterial oxygenation, and fluid loss are also thought to influence central control of exercise intensity (Noakes, 2012). This feedback control loop leads to appropriate adjustments in motor unit recruitment and thus exercise intensity and metabolic activity. An illustration of the feedback, feedforward control of exercise intensity regulation is displayed in Figure 1

Research has focused on investigating the afferent signalling properties of metabolic substrates and by-products, as well as other physiological parameters related to physical exertion. The following section will review the research on the variety of afferent physiological signals thought to influence central control of exercise intensity, before later sections will discuss how maturation may influence the magnitude of these physiological signals.

Metabolic signals

In line with the peripheral model of fatigue, the traditional belief was that during prolonged moderate to high intensity exercise, fatigue was induced when muscle glycogen stores reached a critically low level (e.g. Ahlborg *et al*, 1967). However, more

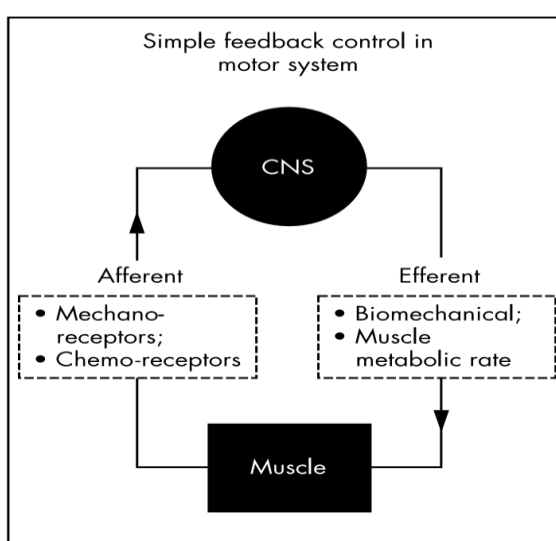


Figure 1. Ulmer's concept of "teleoanticipation", in which afferent signals from various receptors are integrated by the CNS when determining the degree of motor unit recruitment. Source: Lambert *et al* (2005)

recent research has suggested that muscle glycogen content is never completely depleted at exhaustion, whilst reporting a large inter-subject variability in muscle glycogen content at the point of exhaustion (Rauch *et al*, 2005). An alternative school of thought suggests that glycogen has afferent signalling properties to the CNS, influencing feedforward programming of exercise intensity.

In 1998, Weltan and co-workers examined the influence of muscle glycogen content on metabolic regulation during exercise. Participants performed 145 minutes of cycling at 70% of VO_2max with low muscle glycogen and normal muscle glycogen, while euglycemia was maintained through a glucose infusion. The results displayed an increase in fat oxidation both at rest and during exercise, with a lower respiratory exchange ratio

(RER) in the low muscle glycogen trial. This led the authors to the suggestion that the low muscle glycogen levels induced a shift towards lipid metabolism at rest and during exercise due to afferent signals from muscle chemoreceptors to the CNS, which resulted in an increase in circulating norepinephrine concentration, and a concomitant increase in free fatty acid concentration. While the exact mechanism by which muscle glycogen levels are relayed to the CNS are unknown, Weltan *et al* (1998) suggested that changes in the concentration of a glycolytic intermediate may trigger afferent sensory feedback from chemoreceptors in contracting muscles.

Since then, numerous studies have provided support for the notion that glycogen may have afferent metabolic signalling properties (e.g. Lambert *et al*, 2005; Rauch *et al*, 2005; Noakes, 2012). In their 2005 study, Rauch and colleagues examined the pacing strategy and end muscle glycogen content of cyclists performing two hours of cycling at 73% of $\text{VO}_{2\text{max}}$, immediately followed by a one hour time trial. The test was performed under two conditions, once when participants were carbohydrate loaded, and once when they were not carbohydrate loaded. Interestingly, participants started the one hour time trial at a similar workload under both conditions. However, following the first minute of cycling in the unloaded condition, workload significantly decreased in comparison to the loaded trial. This suggests that the pacing strategy was adapted in response to the metabolic signal which informed the CNS that glycogen levels (or the concentration of a glycolytic intermediate) were lower. Furthermore, that the pacing strategy was only adapted following the first minute of cycling in the unloaded condition implies that there may be a delay in afferent signals being sent to the CNS during the initial stages of exercise.

Another significant finding was the large inter-subject variation in muscle glycogen levels at exhaustion. In contrast, intra-subject glycogen levels at exhaustion in the two trials showed very little variability, irrespective of initial glycogen levels. This indicates that large individual differences exist in critical “stopping” muscle glycogen concentrations (Lambert *et al*, 2005). Furthermore, it is well known that metabolic variables such as muscle glycogen levels (or levels of a glycogen intermediate) and lactic acid concentration are relayed through group III and IV afferents to various sites in the CNS, influencing both central motor drive and the cardiovascular and ventilatory reflex response (Weltan *et al*, 1998; Amann *et al*, 2011). Evidence to support this comes from the significantly greater level of muscle fatigue accumulated when group III and IV afferents are blocked, resulting in central motor drive being less inhibited (Amann *et al*,

2011). Despite the increase in central motor drive observed in this study, performance during an incremental cycling test was impaired when the afferents were blocked due to an inhibited cardiovascular and ventilatory response and a concomitant reduction in muscle oxygen transport. This study provides further support for the interpretation that metabolic signals are involved in the regulation of exercise performance, allowing completion of exercise without complete system failure.

Another metabolic by-product which has been suggested to act indirectly as a signalling agent is lactate (Lambert *et al*, 2005; Philp *et al*, 2005). Acid-sensing ion-channels (ASICs) are highly expressed sensory neurons which act through afferent pathways to pain centres in the brain (Immke & McCleskey, 2001). An increase in lactic acid, and concomitant reduction in blood and muscle pH, may stimulate afferent feedback to pain centres in the brain, bringing about reductions in exercise intensity as a protective mechanism, rather than directly causing fatigue through complete system failure.

The “lactate shuttle hypothesis” describes the role of lactate in delivery of oxidative and gluconeogenic substrates, as well as cell signalling (Brooks, 2009). In their review of lactate as a signalling molecule, Philp *et al* (2005) provide support for the Central Governor hypothesis, citing the “growing evidence of lactate utilisation in the brain *via* the astrocyte-neurone lactate shuttle, a system clearly capable of affecting substrate delivery and neurone function”. Taking into account that lactate concentration is elevated during exercise, and reaches maximal levels around the termination of exercise, as well as its afferent signalling properties, it is plausible that lactate plays a role in the central regulation of exercise intensity.

In conclusion, a growing body of evidence proposes that a range of metabolic substrates and by-products may have signalling properties to the CNS, providing information on the metabolic status of peripheral skeletal muscle cells. It is suggested (Swart *et al*, 2012) that these signals are integrated by the CNS, and influence central motor drive and the efferent control of motor unit recruitment to ensure that homeostasis of peripheral physiological systems is maintained within tolerable limits. Given that pre-pubescent children display differences in the metabolic response to high-intensity exercise, such as a lower accumulation and faster removal of metabolic by-products (Ratel *et al*, 2006), this in turn may influence afferent feedback to the CNS, thus affecting central regulation of exercise intensity and pacing strategies. These differences in pacing strategies may be particularly apparent in sports characterised by high-intensity intermittent exercise, such

as association football, where maturation has been found to impact on repeated sprint activity and recovery between bouts of activity (Lazaar *et al*, 2002; Buchheit *et al*, 2010).

Core Temperature

Another example of afferent sensory feedback that is integrated as part of the central regulation of pacing strategies is core temperature. Traditionally, it was believed that exercise performance was impaired in hot conditions because of the need to perfuse the exercising muscles and skin to aid thermoregulation. This would result in a decrease in blood flow and thus oxygen supply to the working muscles (McArdle *et al*, 2006).

More recent research has moved away from this critical core temperature hypothesis. A consistent finding in the literature is that, during self-paced exercise in the heat, power output is reduced before critical core temperatures are reached (Marino *et al*, 2004). For example, Tucker *et al* (2004) examined the effect of temperature on performance during a self-paced 20km cycling time trial. One trial was conducted in hot temperatures (35°C) and one in cool temperatures (15°C). The results showed that power output in hot conditions were reduced during the first 6km of the time trial compared with the cool conditions. Due to this reduced power output, rectal temperature, heart rate and RPE remained similar in both trials. Only during the final kilometre of the trial, when power output and iEMG were increased during the “end spurt”, did rectal temperature become significantly higher in the hot conditions (39.2°C) compared with the cool conditions (38.8°C). Thus, the authors implied that the pacing strategy during the 20km time trial was altered as part of an “anticipatory regulation of performance”, thereby preventing excessive heat storage and premature increases in body temperature.

During prolonged high-intensity intermittent team sports, ambient temperatures have been found to influence pacing strategies. A consistent finding is that when team sports are performed in the heat, there is a significant reduction in low-intensity activity (Duffield *et al*, 2009; Aughey *et al*, 2013). This suggests that players alter their movement patterns in order to ensure that critical core temperatures are not reached, allowing high-intensity activity to be sustained throughout a match through a reduction in low-intensity activity, providing further support for the notion that intensity is centrally regulated to prevent premature fatigue and threats to homeostasis.

Thus, growing evidence implies that the afferent signals sent to the CNS regarding body temperature cause alterations in efferent control of motor unit recruitment and pacing strategies in anticipation of the increased thermal strain, to ensure that critical core temperatures are not reached, thus maintaining internal homeostasis and thermoregulation. While research on thermoregulatory differences related to maturation is inconclusive, some evidence has suggested that children possess physiological and anatomical characteristics that have been linked with impaired thermoregulatory responses to exercise in the heat (Sinclair *et al*, 2007). Therefore, differences in thermoregulatory responses in pre-pubescent athletes may induce alterations in afferent feedback relating to core temperature, thus influencing pacing strategies.

Exercise Duration

Another integral factor in regulating pacing strategies is the duration or distance of a given task. As well as peripheral signals, the CNS also integrates information on the end-point, and distance covered when selecting an appropriate pacing strategy (Faulkner *et al*, 2011). The integration of these cues is dependent on knowledge of the task and previous experience (Micklewright *et al*, 2009).

In their 2008 review, Abbiss and Laursen define a number commonly observed pacing strategies in sport. While a number of unique pacing strategies may exist, four broad categories have been described, including:

- An “all-out” pacing strategy, in which athletes begin a race at maximal pace, and speed gradually declines towards the end of the race.
- An even paced pacing strategy, in which pace is maintained at a similar submaximal level throughout the race.
- A slow start strategy in which individuals begin the race at a submaximal pace and speed up as the race proceeds.
- A parabolic pacing strategy, in which athletes begin fast, slow down for the middle part of the race, and increase their speed towards the end of the race.

The pacing strategy selected depends largely on the length and nature of the race. During events of short duration or distance (i.e. ≤ 30 seconds), it is common for athletes to use an “all-out” or “positive” pacing strategy, with a high initial speed which declines

throughout the exercise bout. For example, during Wingate tests lasting 30 seconds, power output gradually declines to almost 50% of the starting power output by the finish (Ansley *et al*, 2004). In a 12 year analysis of pacing strategies in international swimming competitions, Saavedra *et al* (2012) found that speed was highest at the beginning of the race, and gradually declined towards the end during 100m and 200m individual medley events in elite male swimmers.

During longer exercise bouts (i.e >30 seconds), pacing strategies become more even. Endurance races are often characterised by a fast start, followed by a decline in speed during the middle parts of the race, and an “end-spurt” in which athletes increase their speed in the final stages of the race (Tucker & Noakes, 2009). For example, Tucker *et al* (2004) found that during a 20km self-paced time trial speed was decreased after an initial fast start, with an end-spurt in the final 5% of the trial in recreational male cyclists. The author proposed that the expected exercise duration is integrated by the CNS, and has an important influence in the feedback-feedforward programming of an appropriate pacing strategy. The accuracy of expectations on the duration and/or distance remaining during a task is influenced by knowledge and experience of the task. This was displayed in the work of Foster *et al* (2009), who found that giving participants’ experience of a given task through repeated trials resulted in alterations in pacing strategies, with increased power output during the early and middle parts of the time trials. The authors suggested that the observed differences in pacing strategy were a result of increased confidence that a higher power output could be sustained without a meaningful loss of performance before the finish.

The importance of knowledge of end-point of exercise is demonstrated by numerous studies that have manipulated knowledge of duration and/or distance through deception trials. Billaut *et al* (2011) investigated the influence of prior knowledge of sprint number on pacing during intermittent repeated sprint exercise. Subjects performed repeated six second maximal effort cycle sprints under three conditions: a control trial in which participants were correctly told that they would complete ten sprints, a deception trial in which they were told they would perform five sprints, but upon completion of the fifth sprint were asked to perform a further five sprints, and an unknown trial, in which participants were not told how many sprints they would complete.

The results showed that during the deception trial, the initial work output, EMG activity and accumulated work output was significantly greater than in the control trial and the unknown trial. When participants were not given information on the number of sprints

they would perform, work output was significantly lower than in the control or deception trial. This suggests that pacing occurs even during short duration, “all-out” intermittent type exercise, and is influenced by the knowledge of the number of efforts required. When the duration of exercise is unknown, participants seem to employ a more conservative pacing strategy in order to avoid premature fatigue.

Eston *et al* (2012b) assessed the influence of deception on RPE and heart rate (HR) during 20 minutes of running or cycling. They found that when participants were deceived on the duration of the task, there was an acute increase in RPE once the true requirements were made known despite HR being at a similar level as with a control trial. Furthermore, when participants were not informed of the task duration, RPE remained lower throughout the trial compared with the control. The authors suggested that differences in RPE reflects the disruption to the feedback/feedforward mechanisms of pacing, highlighting the importance of knowledge of end-point when selecting an appropriate strategy.

Perceived Exertion

Another important factor in the regulation of pacing strategies are the subjective perceptions of exertion. Ratings of perceived exertion (RPE) are suggested to be the conscious manifestation of numerous psychological and physiological variables (Hampson *et al*, 2001). The Borg scale (Borg, 1982) has been causally linked to several physiological parameters, such as heart rate, blood lactate, core temperature, muscle glycogen levels and muscular force output (e.g. Baldwin *et al*, 2003). However, it has been argued that the determinants of RPE may be more complex, and a crucial constituent in the regulation of exercise intensity (Tucker, 2009).

During fixed-rate laboratory-based exercise until exhaustion, Noakes (2004) proposes that the rate of RPE increase determines the duration of exercise. That is, as the exercise duration increases, there is an incremental increase in RPE until maximally tolerable values are reached and exercise is terminated. Noakes (2004) and Tucker (2009) further propose that the rate of RPE increase may be pre-set by the subconscious brain, which gathers information regarding the energy status of the body through afferent feedback. The brain then interprets this feedback in the context of the task and the expected duration or duration remaining, and sets the conscious RPE to ensure that maximally tolerable feelings of exertion are reached at the end of exercise, thus optimising performance. For

example, during exercise in a carbohydrate depleted state, the rate of RPE increase has been found to be higher than when carbohydrate levels are normal (Baldwin *et al*, 2013). This may imply that afferent feedback concerning glycogen levels in the carbohydrate depleted state led to the higher rate of RPE increase, in order to ensure that exercise was terminated before catastrophic system failure, which in this case was glycogen depletion.

Tucker (2009) suggests that during self-paced exercise, momentary RPE are a manifestation of the body's homeostatic status, and this momentary RPE is compared with an "expected RPE" based on pre-exercise expectations of the demands of the task. These pre-exercise expectations have been termed the "performance template" (Foster *et al*, 2009). The individual then interprets whether the momentary RPE is sustainable within the context of the task. That is, if during a race an athlete's RPE is perceived as being too high, then power output will decrease to ensure that the race can be finished before premature fatigue and a significant drop in performance occurs. To investigate this, de Koning *et al* (2011) assessed the application of a "hazard score", which includes the product of the momentary RPE multiplied by the fraction of the race distance remaining. The results of the study, which was conducted on runners and cyclists, displayed that the hazard score was able to accurately predicted changes in velocity throughout a race. If participants had a low hazard score, then their velocity would generally increase, whilst the opposite occurred if hazard scores were high. This suggest that subjective perceptions of exertion play an important role in determining momentary changes in power output during exercise.

The pre-programmed performance template described by Tucker (2009) is dependent on knowledge and previous experience of similar tasks. Foster and colleagues (2009) examined the influence of previous experience on the development of optimal pacing strategies for a given task. Participants were split into four groups, and performance was measured during six repeated trials of a 3km cycle time trial (group A), three 2km rowing time trials (group B), four 2km rowing time trials with a training period between trials two and three (group C), and three 10km cycle time trials (Group D). Improvements in performance were found in all of the successive trials, with a 6% improvement over the first three cycle trials, and a 10% improvement after six trials. These improvements in performance were found to be independent of any training effect. Rather, the improved performance throughout the trials was found to be a result of alterations in pacing strategies, with increased power output during the early and middle stages of the race. This increased power output was accompanied by a higher RPE in the early and middle

stages, suggesting that the participants became more confident in their ability to sustain higher levels of RPE without a meaningful slowdown before the end of the trial. Thus, the performance template was adapted throughout the successive trials. Given that there was a continuous improvement in performance throughout the six cycle time trials, this implies that the process of developing an optimal pacing strategy takes more than six trials to be complete.

In summary, conscious perceptions of fatigue and exertion appear to play an important role in the regulation of pacing strategies. Conscious RPE are generated from afferent physiological feedback and these perceptions ensure that an appropriate pacing strategy is selected in the context of a given task through the interpretation of whether momentary RPE is sustainable with the demands of the task in mind, thus preventing premature fatigue and allowing for optimal performance times. In less mature, pre-pubescent athletes, it is possible that differences in the physiological and metabolic responses to high-intensity exercise may lead to a reduced metabolic signal. In turn, this may lead to lower perceptions of physical exertion (Ratel *et al*, 2004), thus influencing pacing strategies.

2.3 - Maturity and Pacing Strategy

Physical maturation from childhood to adulthood is associated with many changes in physical performance, such as strength, power, endurance, fatigability, and recovery from exhaustive exercise (Lloyd & Oliver, 2014). Various factors, such as morphological, metabolic, neuromuscular, hormonal, and cardiovascular changes, can account for the child-adult differences in these performance attributes. These biological changes primarily occur during the adolescent period, which is characterised by physical, sexual and cognitive development. In general, post-pubertal adolescents are stronger, faster and more powerful than pre-pubertal children, largely due to physical advantages in stature and mass (Lloyd & Oliver, 2014).

While little or no research has specifically assessed the influence of maturity on pacing *per se*, the differences in the physiological, metabolic and cardiovascular characteristics of pre-pubescent children and post-pubescent adolescents are likely to have an influential role on the feedback and feedforward regulation of exercise intensity and pacing. Insight into the potential influence of maturity on the distribution of work during exercise can be gained by analysing age/maturational differences in fatigability. Furthermore, an analysis

of running patterns in team sports in relation to maturation may further indicate the potential influence on pacing strategies.

Maturation Influence on Fatigability

Biological maturation during childhood and adolescence has a significant impact on fatigability during prolonged (>15 minutes) and short-duration (<15 minutes) high-intensity exercise. In terms of prolonged exercise, significant natural improvements in endurance are displayed around peak-height-velocity (PHV) (Lloyd & Oliver, 2014). Furthermore, in team sports such as association football and field hockey, post-pubertal adolescents have been found to outperform their younger and/or less-mature counterparts in match running performance, covering more absolute and high-intensity distance (Harley *et al*, 2010). These maturation-related improvements in endurance have been attributed to an improved running economy (Fellmann & Coudert, 1994) and changes to the cardiorespiratory and musculoskeletal system (Philippaerts *et al*, 2006).

While more mature adolescents have many physical advantages over less mature adolescents, a consistent finding throughout the literature is that younger, less-mature adolescents, experience less fatigue during short duration, high-intensity exercise (Ratel *et al*, 2006). For example, while Beneke *et al* (2005) found that peak power output was higher amongst adolescents (mean age 16.3 years) than boys (mean age 12 years), they also noted that the decrement in peak power during the 30 second Wingate test was lower amongst the younger age group. Similarly, Gaul *et al* (1995) compared anaerobic performance between boys and men during a 90 second all-out cycle test, and found that the decrement in power was lower amongst children than men (25.3% vs 32.2%, respectively). Thus, throughout childhood and adolescents, there appears to be a trade-off between the ability to achieve a higher peak power output, and the ability to sustain peak power output.

Furthermore, another consistent finding throughout the literature is that younger, less-mature children/adolescents recover more quickly from high-intensity exercise (Ratel *et al*, 2006). Lazaar *et al* (2002) found that during intermittent repeated sprint exercise, decrements in speed were lower amongst boys (-12%) than in men (-20%). These findings were corroborated by Hebestreit *et al* (1993), who compared the ability to recover from high-intensity exercise in pre-pubertal boys (9-12 years old) and young adults (19-23 years old), and found that pre-pubertal boys needed just 2 minutes to

reproduce 100% of their performance following a Wingate test, while young adults needed 10 minutes.

The rate and magnitude of recovery from high-intensity exercise has been suggested to be maturity-dependent (Ratel *et al*, 2006). An abundance of evidence has displayed that pre-pubertal children display a better ability to resist fatigue during high-intensity intermittent exercise compared with post-pubertal adolescents (Ratel *et al*, 2002; Zafeiridis *et al*, 2005). For example, Dipla *et al* (2009) found that pre-pubescent children displayed greater fatigue resistance than post-pubertal adolescents during high-intensity intermittent exercise. Therefore, during high-intensity exercise, and in athletic competitions characterised by repeated bouts of high-intensity exercise interspersed with recovery, such as in team sports, younger, pre-pubescent children are likely to display a less conservative pacing strategy in terms of their high-intensity activity and recovery durations. This was displayed by Buchheit and co-workers, who, in 2010, analysed the occurrence and nature of repeated sprint sequences in youth association football players from under 13 to under 18 age groups. The results showed that younger age groups displayed more repeated sprint sequences than older, post-pubertal age groups. Furthermore, these repeated sprint sequences contained more and longer sprints than in the older age groups. While the maturational related metabolic and physiological differences are likely to contribute to the differences in pacing strategies observed in team sports, another factor which may influence running patterns is the tactical astuteness of the players. That is, younger players may be less tactically astute, and as a result perform unnecessary work.

Thus, younger, pre-pubescent adolescents appear to display less conservative pacing strategies during relative high-intensity exercise, as a result of better resistance to fatigue and higher rates of recovery. Several potential mechanisms may be responsible for the reduced fatigability in younger athletes. These potential mechanisms will be discussed in relation to pacing regulation in the following sections.

Effect of Age/Maturity on Metabolic Response to Exercise

As discussed earlier, afferent signals from metabolic variables play an important role in the regulation of exercise intensity. Group III and IV afferents relay information on the biochemical status of muscle cells to the hypothalamic locomotor region (Weltan *et al*, 1998). This information effects the feedforward control of muscle recruitment, and the

conscious perceptions of exertion. The magnitude of these metabolic signals influence pacing strategy, as has been displayed by research manipulating the magnitude of these signals (e.g. Rauch *et al*, 2003)

The differences in the metabolic response to exercise between childhood and adulthood have been well-established. A consistent finding in the literature is that children are better equipped to produce energy through aerobic metabolism than through glycolytic pathways compared with adults (e.g. Falgairette *et al*, 1991). This is thought to be related to the higher activity of aerobic enzymes in children (Ratel *et al*, 2006). By taking muscle biopsies at rest, several studies have found greater activity of enzymes within the tricarboxylic acid cycle (Eriksson *et al*, 1972; Berg *et al*, 1986) and beta-oxidation pathway (Haralambie *et al*, 1982) in children and adolescents compared with young adults.

While clear evidence exists suggesting that children have a higher reliance on oxidative metabolism, some research has also suggested that children have a compromised ability to produce energy through anaerobic metabolism. Eriksson *et al* (1972) implied that this ability was dependent on maturational status, basing his findings on a lower concentration of muscle lactate in adolescent boys compared with young adults following maximal exercise, and on the lower activity of glycolytic enzymes such as phosphofructokinase. While other studies have found lower activity of anaerobic enzymes in pre-pubertal children/adolescents (e.g. Berg *et al*, 1986; Kaczor *et al*, 2005), conflicting evidence exists (Haralambie *et al*, 1982). These conflicting results can be attributed to methodological inconsistencies, with studies employing different exercise modes and intensities, differences in subject maturation, size and training status, as well as differences in the unit of measurement. Whilst more research is required, the lower accumulation of metabolic by-products associated with glycolytic metabolism in young adolescents may explain at least some of the variance in recovery between high intensity bouts when compared to adults.

An important mechanism by which children may recover more quickly from high-intensity exercise is their faster resynthesis of phosphocreatine (PCr). Individuals with a high capacity for aerobic metabolism are better able to resynthesise phosphocreatine following high intensity exercise (Bogdanis *et al*, 1996). Kappenstein *et al* (2013) found that PCr resynthesis was faster in children (average age 9.4 years) than in adults (average age 26.1 years) following repeated dynamic planter flexion exercise, and suggested that this was a result of their higher rates of oxidative ATP formation. Furthermore, children

may also have higher rates of muscle lactate and H^+ ion clearance (Ratel *et al*, 2006), which would facilitate faster recovery. Several mechanisms have been suggested to be responsible for this faster removal of metabolic by-products, such as an increased ratio of type 1 muscle fibres (Metaxas *et al*, 2014), which would result in higher rates of lactate oxidation and higher rates of H^+ ions utilisation to produce ATP through aerobic metabolism (Pilegaard *et al*, 1999), as well as faster rates of diffusion of lactate and H^+ ions from muscle to blood (Ratel *et al*, 2006).

Based on the evidence, it is apparent that during maturation, there is a shift in the dominance of metabolic pathways used to provide energy during high intensity exercise specifically. Children and less-mature adolescents rely predominantly on aerobic metabolism as an energy source, with a lesser capacity to produce energy anaerobically which is attenuated through the adolescent growth spurt. This may have important implications when considering the potential influence of maturity on pacing strategies. For example, in team sports, the lower accumulation of metabolic by-products in pre-pubertal adolescents and the concomitantly reduced metabolic signal would allow for younger players to better sustain, recover from and more quickly recommence high-intensity activity (Buchheit *et al*, 2010).

Muscle Composition

Differences in muscle fibre composition may explain some of the variance in recovery from high intensity exercise between adults and children. While limited evidence exists, some studies have suggested that pre-pubescent children have a higher proportion of type 1 muscle fibres. For example, Oertel *et al* (1988) conducted a morphometric analysis of skeletal muscle fibre composition in children, adolescents and adults during autopsy, and found that the proportion of type 1 muscle fibres was 54% at age 6-10 years, to 47% at age 10-15 years, and 42% at age 15-20 years. In a similar study, Lexell *et al* (1992) also found a higher proportion of type 2 fibres in young adults (50%) than in children (35%).

More recently, Metaxas *et al* (2014) assessed muscle fibre composition in youth footballers in three age groups: Group A (11.2 ± 0.4 years old), Group B (13.1 ± 0.5 years old) and Group C (15.2 ± 0.6 years old). The results showed that Group A had a 21% higher distribution of type 1 muscle fibres in comparison with group C. Moreover, the distribution of type 2a fibres was 18% higher in Group C compared with groups A and

B. The authors proposed that the higher distribution of type 2a fibres in the more mature adolescents could be attributed to differences in training programmes, as well as age and/or maturation related increases in fast twitch muscle fibres.

Given that type 2 muscle fibres rely predominantly on anaerobic metabolism to produce energy, a higher proportion of these muscle fibres would lead to an increased accumulation of metabolic by-products. Therefore, the higher proportion of type 1 fibres in pre-pubescent's would lead to a reduced metabolic signal and a more sustainable power output during high-intensity exercise. This may be partly responsible for the less conservative pacing strategies displayed in less-mature adolescents (Buchheit *et al*, 2010). However, given the confounding factors which may be responsible for differences in muscle fibre composition observed, such as differences in the training status of individuals, as well as the limited amount of research in this area, this hypothesis must be interpreted with caution.

Neuromuscular activation may also be related to differences in the regulation of exercise intensity in pre- and post-pubertal adolescents. Studies have suggested that differences in muscle function, such as the increase in peak power and fatigability, can be explained by different levels of motor unit activation (Asai & Aoki, 1996). This was first proposed by Asmussen (1995) who suggested that children's maximal neuromuscular activation was lower than in adults. Dotan *et al* (2012) implied that children have a reduced capacity to recruit glycolytic type 2 motor units compared with adults. Evidence to support this comes from studies applying the interpolated-twitch technique, which is used to estimate the degree of motor-unit activation during volitional effort. During a maximal voluntary contraction, an electrical stimulus is superimposed onto the motor nerve or muscle, and the subsequent superimposed twitch force is measured. The ratio between the volitional contraction and the evoked non-volitional force is used as a measure of the degree of motor unit recruitment. A consistent finding is that children have lower muscle activation than adults (Grosset *et al*, 2008; Paasuke *et al*, 2000).

Mechanoreceptors send information concerning the mechanical load on the muscles to the CNS, influencing the efferent feedforward modulation of force output and performance (Baron *et al*, 2009). Another means by which maturation may influence pacing strategies is through the reduced mechanical load associated with reduced maximal-power generating capacities in pre-pubertal children. Ratel *et al* (2004) found that during high-intensity sprinting or cycling in children and adults, the greater decline in power output in adults was closely related to their higher absolute power output. These

findings were corroborated by Kanehisa *et al* (1995), who found that declines in power output during repetitions of maximal knee extensions in boys and young adults were closely related to the initial absolute muscle power output. The lower muscle mass, as well as the lesser proportion and/or activation of type 2 “fast-twitch” muscle fibres, may reduce the signals sent from mechanoreceptors, thereby influencing pacing strategies.

Therefore, the reduced activation of type 2 muscle fibres in pre-pubertal children is likely to be an important mechanism by which children resist fatigue, and recover more quickly, from high-intensity exercise. A decreased proportion and/or activation of these muscle fibres would lead to differences in the metabolic response to exercise, with a reduction in the accumulation of metabolic by-products. As a result, a reduced mechanical load and metabolic signal would be sent to the CNS, leading to lower RPE and a reduced requirement to lower exercise intensity in order to protect and sustain metabolic systems.

Hemodynamic and Autonomic Responses

Children display a faster rate of recovery of several physiological variables following high-intensity exercise. These include heart rate, VO_2 , cardiac output and ventilation (Ratel *et al*, 2006). The rate of recovery of these variables is likely to be maturation-dependent. For example, Buchheit *et al* (2011) compared hemodynamic recovery from a graded running test to exhaustion in pre-, circum-, and post-PHV youth footballers, and found that heart rate recovery was faster in pre-PHV than circum- and post- PHV players. In support of these findings, Hebestreit *et al* (1993) noted faster recovery of HR, ventilation, VCO_2 and VO_2 in children compared with young adults.

Several mechanisms are likely to be responsible for the faster readjustment of cardiopulmonary variables in pre-pubertal children. Namely, their greater reliance on oxidative metabolism and lower accumulation of metabolites associated with anaerobic metabolism, which in addition to a lower muscle mass, leads to a reduction in the muscle metaboreflex and attenuation of sympathetic activity (Buchheit *et al*, 2011). A decreased reliance on anaerobic metabolism would also lead to a reduced oxygen debt, facilitating a faster HR recovery following high-intensity exercise (Ratel *et al*, 2006). Furthermore, the lower absolute work rate, as well as the greater parasympathetic efferent nervous activity to the heart in pre-pubertal children may also be related to the faster readjustment of cardiopulmonary variables (Buchheit *et al*, 2010).

Given that pacing is influenced by afferent feedback from cardiopulmonary parameters, such as ventilation, arterial O₂ and CO₂ (St. Claire Gibson & Noakes, 2004), this may be another potential mechanism by which maturity influences the central regulation of exercise intensity. For example, during team sports characterised by high-intensity intermittent activity, such as association football, a faster readjustment of heart rate and ventilation may allow for less mature players to sustain, reproduce and recover more quickly from high-intensity phases of play.

Perceived Exertion

Perceived exertion during exercise represents a complex psychophysiological process that integrates several exertional symptoms, such as heart rate, VO₂ and ventilatory activity, which are termed central signals, and metabolic acidosis and sensations from contracting muscles, which are termed peripheral signals (Ratel *et al*, 2006). As previously discussed, RPE has been suggested to work in an anticipatory manner, in which RPE reaches maximal values before catastrophic system failure occurs (Tucker, 2009). In addition, the RPE plays a role in the regulation of pacing strategies through adjustments in exercise intensity when the conscious RPE differs from the “template” or “expected” RPE, derived from previous experience (de Koning *et al*, 2011).

Given that both central and peripheral signals are likely to be lower in magnitude amongst pre-pubertal adolescents (Ratel *et al*, 2006), this would suggest that RPE would be concomitantly lower amongst children. Indeed, several studies have found this to be the case (Bar-Or & Ward, 1989; Ratel *et al*, 2004) with children rating perceived exertion lower than adults at a given relative exercise intensity, particularly during short-duration, high-intensity exercise. For example, Ratel *et al* (2002) found that during intermittent, high-intensity exercise, in which both children and adults performed 10-seconds of sprint exercise, interspersed with 15-seconds recovery, RPE was significantly higher in adults than in children, despite exercise being at the same relative intensity. Thus, the lower RPE at a given exercise intensity are consistent with the lower levels of metabolic parameters in pre-pubertal children. When taking into account the importance of conscious perceptions of exertion in the regulation of pacing strategies (de Koning *et al*, 2011), this highlights the potential influence that stage of maturation may have on pacing.

Cognitive Development

While the maturational related differences in the physiological and metabolic responses to exercise may impact on the sensory-perceptual elements of exercise intensity regulation, i.e. the afferent feedback components; the cognitive, decision making processes involved in the regulation of pacing strategies are likely to be influenced by stage of cognitive development. The ability to subjectively interpret internal physiological signals and rate perceived exertion accurately is a psychological function, influenced by cognition, memory, and understanding of the task (Easton, 2009). As discussed earlier, RPE plays an important role in the decision-making processes involved in pacing, with “momentary” RPE being compared with a “template” RPE, and effecting pace accordingly (Tucker 2009; Easton 2012b). This process relies on the ability to recall memories from previous experience whilst accurately interpreting sensations in real-time, both of which are likely to be compromised by inexperience and lower levels of cognitive maturation (Easton, 2009).

Cognitive processes involved in pacing strategies include planning, anticipation, and logical reasoning (Micklewright *et al*, 2011). These processes are said to develop during the pre-operational (4-7 years of age) and concrete operational (8-12 years of age) period, according to Piaget’s (1972) stages of cognitive development. Furthermore, anticipating the demands of an exercise task requires the ability to form internal spatial representations of a given task, an ability which is said to develop during the concrete operational period (Paiget, 1972; Chinnasamy *et al*, 2012).

Micklewright *et al* (2011) assessed the influence of the stage of cognitive development on pacing strategies during a best-effort, 450m to 900m (depending on age) running task. Schoolchildren were divided into four groups depending on their stage of cognitive development, including: pre-operational (4-7 years old), early concrete operational (8-10 years old), late concrete operational (10-12 years old), and the formal intelligence period (13-18 years old). The results showed that schoolchildren in the pre-operational and early concrete operational period displayed an inability to anticipate exercise demands and appropriately pace efforts, as characterised by a positive pacing strategy, in which speed gradually declined throughout the race. In contrast, those in the late concrete operational and formal intelligence period displayed a more conservative, parabolic-shaped pacing curve. This led the authors to the conclusion that anticipatory pacing strategies are related to age and cognitive maturation. Thus, these results suggest that the stage of intellectual development is likely to influence the cognitive processes involved in making judgements

about the physiological and metabolic demands of a task weighed against one's own physiological and metabolic capacity.

2.4 - Other Factors which Influence Pacing Strategies

Level of Experience

Research has suggested that pacing strategies may be regulated by “pacing schemas” in the brain, which are derived from previous experience of completing fatiguing tasks (Lambrick *et al*, 2013; Waldron & Highton, 2014). Micklewright *et al* (2009) proposed that previous experience influences pacing strategies through alterations in beliefs in performance capabilities, and adjustments in the interpretation of physiological responses. That is, through experience, perceptions of exertion can be influenced independent of changes in fitness, causing differences in conscious pacing decisions, as well as subconscious control of efferent motor unit recruitment.

Foster and colleagues (2009) demonstrated the influence of previous experience on the development of a “performance template” during continuous short-duration high-intensity exercise. Participants in the study repeated self-paced time trials in rowing and cycling, separated by at least 48 hours of rest. During the initial trials, RPE increased at a slower rate, and final RPE was lower than in the later trials. Furthermore, a consistent finding in the study was that the improved performance found in all trials was associated with a more aggressive starting pace. This indicates that, as participants became more experienced in the task, they became more confident that an increase in the initial pace would not result in premature fatigue, and thus the “template RPE” was increased. The change in adopted pacing strategy by the participants highlights the learning effect during successive performance of self-paced exercise.

Moreover, St. Claire Gibson *et al* (2006) suggested that pacing strategies are influenced by the presence of an internal time keeping mechanism. This “internal clock” gives an accurate interpretation of the time and distance that has passed during an exercise bout, allowing for appropriate decisions to be made regarding the pacing strategy employed. They further propose that previous experience may influence pacing strategies through an improvement in the accuracy of this internal clock, by drawing upon memories of previous experience of being in similar situations.

Performance and Fitness Levels

Given that there is a learned element to the adoption of pacing strategies, it may be that an athlete's performance level influences the pattern of work output during exercise. That is, better performing athletes employ a faster start with less of a decline in speed, or have a greater capacity to produce a faster "end spurt" towards the final stages of a race. To investigate this, Renfree and St. Claire Gibson (2013) examined the influence of different performance levels on pacing strategies during the 2009 IAAF Women's World Championship marathon race. Athletes were split into four performance groups based on their finishing times, comprising the first, second, third and fourth 25% of finishers. Results showed differences in the pacing strategies in relation to the performance group. Athletes from groups 2 to 4 selected unsustainable initial speeds, as characterised by significant reductions in speed following a fast start. This resulted in a "positive" pacing profile, in which the second half of the race was slower than the first. In contrast, group 1, the top 25% of finishers, ran at lower relative speeds during the initial stages of the race in comparison with the other groups, allowing them to maintain a higher average speed for the duration of the race. In this case, the better performers displayed a more even pacing strategy, while the lesser performers displayed a gradual decline in speed.

Lima-Silva *et al* (2010) examined the influence of performance levels and physiological variables on pacing strategies in endurance runners. The study had participants complete a self-paced 10km endurance run, before splitting them into a low-performing group and a high-performing group based on their results. Before the endurance race, participants performed an incremental running test to exhaustion on a treadmill to determine VO_2max , lactate threshold and peak treadmill running velocity, as well as three 6-minute bouts at 9, 12 and 15km h^{-1} to determine running economy. The split times during the 10km race were analysed to assess differences in pacing strategies adopted in the low- and high-performing group. The results showed that those in the high-performance group displayed a higher velocity during the first 400m of the race, and this velocity was higher than the average race velocity. In contrast, the low-performance group displayed a significantly lower velocity over the first 400m in comparison with the high-performance group, and this starting velocity was not higher than the average race velocity. The start, middle and end velocities measured during the race were significantly correlated with running economy, lactate threshold and peak treadmill running velocity. These results suggest that performance levels influence the adopted pacing strategy during self-paced,

endurance running activity, and that running economy, lactate threshold and peak treadmill running velocity may be important determinants of the chosen pacing strategy.

More recently, Johnstone *et al* (2015) assessed the influence of performance and fitness levels on pacing strategies during a junior team sport tournament, consisting of five games in four days. The movement patterns across successive games were analysed in participants from a high-standard and low-standard team, while players were also split into a high and low fitness group based on results from an intermittent Yo-Yo recovery test. The results showed that high-standard players began the tournament at a higher intensity compared with the low-standard players, with more high- and moderate-intensity activity. Furthermore, high-standard/high-fitness players were able to maintain this intensity throughout the tournament, whereas high-standard/low-fitness players displayed reductions in intensity throughout the tournament, most likely due to the accumulation of fatigue during successive games. In contrast, low-standard players displayed a negative pacing strategy throughout the tournament, in which they produced an “end spurt” in the latter games. These results highlight the importance a well-developed endurance capacity when determining the pacing strategies, particularly during tournaments in which players are required to compete on consecutive days.

2.5 - Conclusions

The regulation and distribution of exercise intensity, termed pacing strategies, is influenced by several psychophysiological and external factors. Afferent feedback to the CNS from physiological and metabolic systems influence the efferent feedforward control of muscle recruitment, and thus exercise intensity. This feedback also influences subjective sensations of exertion, impacting the conscious decision-making processes involved in pacing of efforts in the context of a given task. The magnitude of peripheral signals, such as metabolic acidosis, and central signals such as ventilation, influences exercise intensity regulation in an anticipatory manner, such that maximally tolerable limits of these variables are not reached before the end of the exercise bout. Thus, external factors which influence these signals, such as temperature or hypoxia, will influence pacing strategies to ensure that homeostasis is maintained within tolerable limits.

While little research has focused on the influence of physical maturation on pacing strategies, the physiological and metabolic changes that occur in response to exercise throughout maturation have been well established (Ratel *et al*, 2006). In general, pre-

pubertal children/adolescents have been found to have lesser absolute power generating capacities than post-pubertal adolescents, but have an enhanced ability to sustain exercise at a higher relative exercise intensity than post-pubertal adolescents. Several factors have been suggested to be responsible for this, such as a reduced metabolic signal during exercise as a result of a predominantly aerobic energy provision, with less contribution from anaerobic metabolism leading to lesser concentrations of metabolic by-products, with concomitantly lower RPE. Furthermore, structural and neuromuscular differences may also play a role in the differences in the exercise response between less and more mature adolescents, particularly during high-intensity exercise.

Given the importance of physiological and metabolic signals in pacing regulation, physical maturation is likely to have an influential effect on pacing strategies. This can be displayed by the differences in running patterns displayed by younger, less-mature athletes in team sports, who have been found to perform at a higher relative intensity than older, more-mature athletes (Buchheit & Mendez-Villanueva, 2014), whilst also displaying more repeated sprint sequences (Buchheit *et al*, 2010). While several studies have found this to be the case, more research is required to specifically assess how stage of maturation influences pacing strategies during self-paced exercise.

Chapter 3 – Study One – Evaluation of Performance and the Influence of Maturation during a Self-regulated Repeated Sprint Task in Elite Youth Association Football Players

3.1 - Introduction

Repeated sprint exercise (RSE) is characterised by short durations sprints (≤ 6 seconds) interspersed with short recovery periods (≤ 60 seconds) (Buchheit *et al*, 2013). This form of training is common across team sports such as association football due to the numerous physiological and biomechanical adaptations associated with repeated sprint training deemed highly relevant to team sport performance (Taylor *et al*, 2015). As such, research has focused on strategies to optimise repeated sprint training through manipulation of variables such as sprint distance and work-to-rest ratios (Little & Williams, 2007), number of sprints (Gharbi *et al*, 2014) and recovery modality (Castagna *et al*, 2008). An important consideration when implementing this training modality, however, is the individual differences in the capacity to recover between sprints in order to reproduce maximal sprint performance, which is predominantly dependent on an individual's oxidative capacity (Glaister *et al*, 2005). Edwards *et al* (2011) suggested that insufficient or excessive recovery periods may lead to a suboptimal training stimulus, and training outcomes may thus be compromised. However, the most common model of repeated sprint training emerging from research in team sports is to use standardised recovery periods or work-to-rest ratios (Taylor *et al*, 2015), which may not provide an optimal training stimulus for each player, particularly when training is performed as a group.

A practical approach that has been suggested to account for individual differences is to allow subjects to choose their own recovery duration based on individual perceptions of recovery (Phillips *et al*, 2014). Recent studies have employed this method in adults and found that subjects could reliably and accurately self-govern recovery periods to maintain a consistent performance during successive sprints (Glaister *et al*, 2010). However, studies investigating RSE with self-guided recovery have been conducted exclusively on adults, with no research investigating the effect of this mode of recovery on performance in young athletes.

Using this self-regulated approach to repeated sprint training requires participants to accurately perceive afferent physiological feedback from the periphery (e.g.

blood/muscle pH, respiratory signals, substrate depletion, metabolite accumulation) to the central nervous system (CNS) within the context of the trial – a process known as teleoanticipation (Ulmer, 1996). The ability to accurately perceive physiological feedback and, in turn, readiness for recommencement of sprint exercise during each recovery interval is also likely to be influenced by psychological factors, such as level of experience (Foster *et al*, 2009) and cognitive development (Micklewright *et al*, 2011).

Taking into account the physiological mechanisms that are suggested to influence the regulation of self-guided recovery intervals, it is conceivable that young athletes at varying stages of maturation may display differences in chosen recovery intervals due to maturation-related differences in the physiological responses to RSE between children and adolescents (Ratel *et al*, 2006; Micklewright *et al*, 2011). For example, pre-pubescent youngsters have been found to recover more quickly during RSE due to a number of suggested mechanisms, such as a higher reliance on aerobic metabolism and lower accumulation and/or faster clearance of metabolites associated with anaerobic metabolism thought to limit repeated sprint performance (Ratel *et al*, 2002; Spencer *et al*, 2005). However, given that the ability to self-govern recovery intervals is also likely to be influenced by cognitive factors, differences in the level of cognitive development and/or level of experience between more and less mature players may influence the ability to self-determine recovery intervals in order to maintain performance during RSE with self-guided recovery. While Micklewright *et al* (2011) found that children in the late concrete operational period (10-12 years old) according to Piaget's (1972) stages of cognitive development model displayed the ability to regulate intensity during a one-off 450-900m run, the influence of maturation on the ability to regulate recovery during RSE is yet to be explored. Information on this area would potentially allow practitioners to make more informed decision making in practice, whilst also giving insight into the influence of maturation on the regulation of intensity and recovery during self-regulated RSE. Moreover, using this teleoanticipatory approach to RSE may promote greater self-awareness of physical capabilities amongst young athletes, which in turn may facilitate improved performance in a competitive setting (Edwards *et al*, 2011).

Furthermore, Edwards *et al* (2011) suggested that using scalar methodologies during self-guided recovery periods may provide an effective practical means of accurately judging the required recovery time in order to maintain performance, and proposed that the applicability of such methodologies should be assessed during other modes interval training. However, no studies have assessed the applicability of this method during RSE.

Thus, the aim of the present study is twofold: 1) To assess the effect of using self-guided recovery periods on performance during a repeated sprint task with and without the use of scalar methodologies to facilitate self-determination of recovery periods; 2) To evaluate the influence of maturation on performance and the ability to self-govern recovery periods during RSE in elite youth association football players.

3.2 - Methods

Participants

Twenty-eight male elite youth association football players (aged 14 ± 0.5 years) from a Scottish professional football academy were selected to take part in the study. Physical characteristics of the participants are presented in Table 1.1. Participants were habituated with RSE and trained three to four times per week in addition to at least one competitive match. Each participant was informed of the study procedures; players and where appropriate, their guardian(s), gave informed written consent prior to data collection. Participants were asked to restrain from physical activity in the 24 hours prior to the repeated sprint trials. The study received institutional ethics approval and conformed to the declaration of Helsinki.

Experimental Design

Baseline data was collected for sprint speed (30 m), body mass, stature, and maturity offset (Table 1.1). Age at peak-height-velocity (PHV), a somatic biological maturity indicator which reflects the maximum velocity in statural growth during adolescence, was used as a relative indicator of maturation as described by Mirwald *et al* (2002). Participants were allocated into a “more” and “less” mature group based on their stage of growth using a median split, with the less mature group 1.2 ± 0.5 years pre-PHV and the more mature group 0.9 ± 0.6 years post-PHV (Table 1).

Prior to data collection participants were habituated with the repeated sprint protocol and readiness (PR) scale (Table 1.2) which has been used elsewhere to self-guide recovery durations (Edwards *et al*, 2011).

Prior to the repeated sprint protocols participants were fitted with a Polar HR monitor (H7 HR monitor, Polar Electro, Finland), before completing a self-selected five minute warm-up. Each protocol consisted of 10 x 30 m sprints interspersed with passive recovery periods under three experimental conditions, with participants performing the sprints individually and each trial observed by the researcher alone. Participants began each sprint from a standing start 0.5 m behind the timing gate. Following each sprint, participants were instructed to decelerate and walk back to the starting line before performing the subsequent sprint. Sprint and recovery times were measured using infrared timing gates (SmartSpeed, Fusion Sport, Australia). The experimental conditions were as follows:

- 1) Perceived readiness trial (PR). Participants used a perceived readiness scale to guide recovery duration between each of the ten sprints. With the exception of the first sprint, all other efforts were initiated by the participant with no input from the investigators. Players were instructed to self-guide their own recovery and attempt to use the minimal amount of recovery time required to maintain performance in each sprint. To attempt to facilitate the participants in interpreting physiological feedback and recommencing sprint exercise once they were adequately recovered in order to maintain sprint performance, a perceived readiness (PR) scale of 1-7 was put in front of the players during inter-interval recovery periods (Table 2). Participants were instructed to perceive what number they felt best described their perception of exertion, and to recommence sprint exercise once they reached ‘four’ on the scale (adequately recovered). This was defined as the minimal amount of recovery required to perform their fastest possible 30 m sprint.
- 2) No external cue (NEC). Similar to the PR trial, participants were given the freedom to self-regulate recovery intervals and were instructed to attempt to use the minimal amount of recovery time required to reproduce their fastest 30 m sprint. However, during this trial, participants were given no external guidance to assist them in self-determining their recovery duration. This trial was used to indicate whether using a PR scale to guide recovery had any effect on the ability to accurately self-regulate recovery intervals.
- 3) Standardised recovery (SR). During this trial, all participants were given 30 seconds of recovery between sprints, as in the Castagna *et al* (2008) study.

All three RSTs were completed in a randomised order, with participants blinded to each experimental condition until after the completion of the warm-up. Three weeks were given between each of the three trials. All sprints were performed on an indoor synthetic pitch, and trials were performed at the same time of day to minimise the influence of circadian rhythm on physiological and psychological functions.

Outcome measures

Repeated sprint performance was measured through percentage sprint decrement ($100 \times (\text{total sprint time} \div \text{ideal sprint time (i.e. best sprint time} \times \text{number of sprints)}) - 100$) (Glaister *et al*, 2008) and average sprint time. During the PR and NEC trials, inter-interval recovery durations were recorded and the intra-subject variation in recovery

duration during each condition was assessed using a within-trial coefficient of variation (CV). Mean HR ($b \cdot \text{min}^{-1}$) was used to indicate the physiological demand of the trials.

Statistical analysis

Analyses were performed using SPSS (IBM, version 22). Independent sample t-tests were used to assess differences in baseline variables between the groups. Levene's test of homogeneity of variance was performed to ensure normality of the population for every dependent variable. With the assumption of normality confirmed, a two-way repeated measures ANOVA (group x trial) with Bonferroni corrections was used to analyse within-subject and between-group differences in percentage sprint decrement, mean sprint time, mean recovery duration, recovery variation and mean heart rate during the three trials. This analysis was conducted in order to determine within-subject effects for all subjects, as well as for the more and less mature groups individually. In the event of a significant main effect or trial x group interaction, Bonferroni *post-hoc* testing was performed. Repeated measures ANOVA with Bonferroni corrections was used to assess changes in sprint time and recovery duration within each trial. Cohens *d* effect sizes (ES) were used to quantify the magnitude of mean differences between subjects and between trials (*trivial* = $d \leq 0.1$; *small* = $d > 0.2, < 0.5$; *moderate* = $d > 0.5, < 0.8$; *large* = ≥ 0.8) (Cohen, 1992). Statistical significance was accepted at $p < 0.05$. All results are expressed as mean \pm SD unless otherwise stated.

3.3 - Results

Comparison of repeated sprint trials

The average sprint times for all participants during the PR, NEC and SR trials are displayed in Figure 1.1a. A significant main effect for trial on percentage sprint decrement was observed ($F_{2,27} = 7.613$; $p < 0.01$), with *post-hoc* comparisons displaying a significantly higher percentage sprint decrement during the NEC ($p < 0.01$; $ES = .82$) and PR trial ($p < 0.05$; $ES = .57$) compared with the SR trial (Table 1.3). No significant differences were found between percentage sprint decrement during the PR and NEC trials ($ES = .57$). A significant main effect for trial on average sprint time was found ($F_{2,27} = 10.618$; $p < 0.01$), with *post-hoc* comparisons revealing a significantly longer average sprint time during the PR ($p < 0.05$; $ES = .39$) and NEC trial ($p < 0.05$; $ES = .39$) compared with the SR trial. No significant differences were found between mean sprint times in the PR and NEC trials ($ES = .21$; Table 1.3).

A significant main effect for trial on average recovery duration was found ($F_{1,27} = 32.506$; $p < 0.01$), with average recovery duration significantly longer during the SR trial compared with the PR ($ES = 1.88$) and NEC ($ES = 2.05$) trials (both $p < 0.01$). No significant differences were observed between the average recovery duration taken during the PR and NEC trials ($ES = .21$; Table 1.3). Within-subject variation in recovery duration (CV) was not significantly different between the two trials ($ES = .21$; Table 1.3).

A significant main effect for trial on mean HR was found ($F_{2,27} = 32.506$; $p < 0.01$), with *post-hoc* comparisons displaying significantly higher mean HR during the PR ($p < 0.05$; $ES = .65$) and NEC trials ($p < 0.01$; $ES = .67$) compared with the SR trial. No differences were found between mean HR during the PR and NEC trials ($ES = .05$; Table 1.3).

Influence of maturation

Baseline differences in physical characteristics between the groups are displayed in Table 1.1. The more mature group had a significantly shorter 30 m sprint max time than the less mature group ($p < 0.01$).

No significant maturity x trial interaction was found for sprint decrement ($F_{2,52} = 1.929$; $p > 0.05$), recovery duration ($F_{1,52} = 1.052$; $p > 0.05$), mean HR ($F_{2,26} = 1.973$; $p > 0.05$) or recovery CV ($F_{2,52} = 1.139$; $p > 0.05$).

Table 1.4 displays all within-group and between-group comparisons in performance, recovery and physiological variables in all of the three trials. Within-group comparisons

showed that the more mature group displayed a significantly longer average sprint time during the NEC trial compared with the SR trial ($p < 0.05$; $ES = .51$). There were no significant differences in percentage sprint decrement in any of the three trials in the more mature group (PR vs SR $ES = .31$; NEC vs SR $ES = .39$; PR vs NEC $ES = .15$). The less mature group displayed a significantly higher percentage sprint decrement in the NEC ($p < 0.05$; $ES = 1.31$) and PR trial ($p < 0.05$; $ES = .83$) compared with the SR trial. No significant difference was found between percentage sprint decrement in the PR and NEC trials ($ES = .76$). Furthermore, the less mature group also displayed a significantly longer mean sprint time during the PR ($p < 0.05$; $ES = .5$) and NEC ($p < 0.05$; $ES = .96$) trial compared with the SR trial. Average sprint times for the more and less mature group are displayed in Figure 1.1b and 1.1c, respectively, whilst individual differences in percentage decrement between the three trials are displayed in Figure 1.2.

Between group comparison revealed no significant differences between percentage sprint decrement during the SR trial ($ES = .56$; Table 1.4). In contrast, the less mature group tended to show a higher sprint decrement during the NEC trial ($ES = .51$; Table 1.4), although differences were not statistically significant. The average recovery duration employed by the less mature group during the PR and NEC trials tended to be shorter than the more mature group, (PR $ES = .52$; NEC $ES = .55$; Table 1.4), with the less mature group using a consistently shorter recovery period during both trials (Figure 1.4). No significant differences were found in HR between the more and less mature group during any trial (PR $ES = .38$; NEC $ES = .10$; SR $ES = .19$).

3.4 - Discussion

The primary aim of the present study was to assess performance during a 10 x 30 m repeated sprint trial with either self-guided recovery using a PR scale, with NEC, or using a 30-second SR period in young elite association football players. The results of the study suggest that using these different recovery modalities has a significant impact on performance, with participants displaying significantly more fatigue and higher average sprint times during the self-guided recovery trials when compared with a traditional SR period. Previous studies investigating responses in adult populations have suggested that using self-regulated recovery periods may be a useful alternative during RSE when maintenance of work is desired, whilst also providing an individualised approach to repeated sprint training (Phillips *et al*, 2014). However, while Phillips *et al* (2014) found that participants displayed a tendency to *overestimate* the recovery time required in order to maintain performance during repeated sprint cycling - as displayed by a maintenance in performance when self-selected recovery periods were reduced by 10% - the results of the present study suggest that adolescents have a tendency to *underestimate* the recovery time required in order to maintain performance.

During the PR and NEC trials, both percentage sprint decrement and average sprint time were significantly higher than during the SR trial (Table 1.3). Analysis of the sprint times revealed significant decrements from the first sprint by the third sprint in the PR and NEC trials (Figure 1.1a). In contrast, sprint time during the SR trial became significantly slower than the first sprint at sprint five (Figure 1.1a), showing a delay in the fatigue response. Furthermore, that the average recovery durations taking during the PR and NEC trial were significantly shorter than the 30 seconds provided in the SR trial (Table 1.3), during which performance was better maintained, lends support to the assertion that participants in the study underestimated the amount of recovery time required to maintain performance.

Previous research has displayed that using shorter recovery intervals between sprints results in higher physiological stress and fatigue during RSE (Balsom *et al*, 1992). The present study found that average HR was significantly higher during the PR and NEC trials compared with the SR trial (Table 1.3), likely due to the shorter recovery intervals causing incomplete recovery between sprints, resulting in a higher degree of physiological stress. During RSE, the contribution of PCr hydrolysis and anaerobic glycolysis towards ATP resynthesis gradually decreases during successive sprints, with a concomitant increase in aerobic metabolism (Girard *et al*, 2011). This shift in energy

system metabolism results in compromised performance during successive sprints due to a reduced capacity to resynthesize ATP at the required rate. In order to limit fatigue during RSE, sufficient recovery must be taken given in order to allow restoration of PCr stores and removal of metabolic by-products associated with fatigue and decrements in performance (Spencer *et al*, 2005). Given the higher fatigue and shorter recovery intervals displayed during the PR and NEC trials, it is plausible that the recovery intervals allowed for incomplete recovery of these systems. However, this suggestion should be aired with caution, as physiological data in the present study was limited to average HR. Closer analysis of the heart rate and blood lactate responses to RSE with self-guided recovery may give a clearer indication of the physiological and metabolic mechanisms responsible for the higher degree of fatigue displayed during trials with self-guided recovery. This in turn may allow for more informed decision making for practitioners considering implementing this form of training in young athletes through a better understand of the energy systems being utilised during self-regulated RSE, thus allowing for specific adaptations to be targeted.

To facilitate the players in self-determining recovery intervals with the aim of maintaining performance throughout the sprints, the study implemented a PR scale (Edwards *et al*, 2011), and compared the performance and recovery variables to that of the NEC trial. While Edwards *et al* (2011) suggested that using scalar methodologies may provide a practical means of facilitating self-guidance of recovery intervals, the present study found no significant differences between the average recovery time taken between sprints in the PR or NEC trial (Figure 1.3). Furthermore, no significant differences in performance variables were observed between the two trials, suggesting that using scalar methodologies to assist participants in determining the appropriate recovery periods may be less effective in young athletes.

A secondary aim of the study was to assess the influence of maturation on performance and recovery variables, and the ability to determine recovery intervals during RSE with self-guided recovery. When performance between maturation groups was compared, sprint decrement in the SR trial was *moderately* higher in the more mature group (Table 1.4). Thus, when participants were given the same amount of recovery time, the less mature group displayed a better ability limit the decline in performance throughout the sprints. From a physiological perspective, this implies that the less mature group may have been better equipped to resist fatigue during RSE. Previous research has similarly suggested that the ability to resist fatigue during RSE may be maturation-dependent

(Ratel *et al*, 2006), with pre-pubescent children displaying less fatigue and/or a propensity for recovery following high-intensity exercise compared with post-pubescent adolescents (Buchheit *et al*, 2011). For example, during 10 second cycle sprints interspersed by 30 seconds of recovery, Ratel *et al* (2002) found that pre-pubescent children displayed a lower reduction in peak-power output compared with post-pubescent adolescents. Mechanisms proposed to be responsible for the attenuation of fatigue during RSE in children include metabolic factors, such as a higher reliance on aerobic metabolism and a lower reliance on anaerobic glycolysis, leading to a reduced accumulation of metabolic by-products thought to induce fatigue (Eriksson *et al*, 1971; Girard *et al*, 2011), hemodynamic and autonomic factors, such as faster parasympathetic reactivation and heart rate recovery (Buchheit *et al*, 2011), morphological factors, such as a reduced ratio of type 2 muscle fibres (Metexas *et al*, 2014), and neuromuscular factors, such as a lower level of volitional activation of high threshold (type 2) motor units (Dotan *et al*, 2012). This in turn may lead to a reduced metabolic-signal and lower perceptions of exertion during RSE (Ratel *et al*, 2004). Furthermore, an important determinant of fatigue during RSE is the initial sprint time, with an inverse correlation consistently found between initial sprint time and percentage decrement (Girard *et al*, 2011). Given that participants in the more mature group were significantly faster than their less mature counterparts (Table 1.1), this may further explain the higher degree of fatigue that appeared to occur during the SR trial in the more mature participants.

However, despite their enhanced ability to resist fatigue when a SR period was given, the less mature group displayed *moderately* higher percentage decrement compared with the more mature group during the NEC trial (Table 1.4). While both the more and less mature group displayed the highest degree of fatigue during the NEC trial compared with the other trials (Table 1.4), this mode of recovery had a more detrimental impact on performance in the less mature group. Moreover, Figure 1.4 displays that the less-mature players consistently used a shorter recovery period between the sprints during the NEC trial compared with the more mature group. This suggests that while less mature players may physiologically be better equipped to resist fatigue during RSE, they display an impaired ability to accurately interpret this when self-guided recovery was used with NEC and show more of a tendency to underestimate the required recovery time, resulting in a higher decrement in sprint speed.

The ability to subjectively interpret internal physiological signals and accurately perceive readiness for recommencing high intensity exercise in the context of the task is a

psychological function, influenced by cognition, previous experience, and understanding of the task (Eston, 2009). This process relies on the ability to recall memories from previous experience whilst accurately interpreting sensations in real-time, both of which are likely to be compromised by inexperience and lower levels of cognitive maturation (Micklewright *et al*, 2009). Cognitive processes involved in the determination of recovery periods include planning, anticipation, logical reasoning, and the ability to form internal spatial representations of a given task (Micklewright *et al*, 2011; Chinnasamy *et al*, 2012). Thus, it could be speculated that the more inaccurate recovery durations taken by the less-mature group during the NEC trial were a result of reduced intellectual development, understanding of the task, and/or a lower level of experience.

Although participants in the present study regularly participate in RSE, one limitation was that only one habituation session on RSE with self-regulated recovery was given. Glaister *et al* (2010) found that the ability to maintain sprint performance with self-selected recovery periods improved across four repeated trials, indicating that this may be a learned process. Furthermore, Foster *et al* (2009) suggested that the pattern of learning the “performance template” is a process that takes several trials. Thus, if players were given more habituation sessions, it is possible that their ability to accurately interpret physiological signals and self-determine recovery duration in the context of the task may have improved.

In conclusion, using self-guided recovery intervals between repeated sprints has a significant impact on performance when compared with a traditional SR in elite youth association football players. Specifically, players underestimate the recovery duration required in order to maintain performance throughout the sprints, resulting in a higher degree of physiological stress and fatigue. The detrimental impact on performance when using self-guided recovery with NEC appears to be more pronounced in less mature players, who used a consistently shorter recovery period than their more mature counterparts whilst also displaying a higher degree of fatigue, suggesting that less mature players have an impaired ability to interpret physical capabilities in the context of the task.

Practical Applications and Future Research

This study has demonstrated that elite youth football players underestimate the recovery duration required to maintain performance during a repeated sprint task with self-guided

recovery. Therefore, when maintenance of sprint performance is desired, implementing self-guided recovery in between sprints in young football players may not be an effective training tool. However, given that previous studies have found that the ability to accurately self-regulate recovery is a learned process (Glaister *et al*, 2010), and that participants in the current study underwent only one habituation session, the notion that this ability may be improved with repeated trials cannot be dismissed, and this presents an area for future research. If players are able to improve their ability to accurately interpret physiological signals within the context of the trial in order to maintain performance, then this may represent an effective training tool in terms of individualising repeated sprint training as well as facilitating greater self-awareness of physical capabilities amongst young players (Edwards *et al*, 2011), both of which may lead to improved performance in a competitive setting. Furthermore, the use of scalar methodologies to facilitate self-determination of recovery periods appears to be an effective means of improving performance and coaches should consider its implementation when conducting this form of training.

Tables

Table 1.1. Physical characteristics of both groups combined, and the less mature and more mature group.

Variable	Pooled characteristics	Less mature group	More mature group
	n=28	n=14	n=14
Age (years)	13 ± 0.9	12 ± 0.4 *	14 ± 0.5
Height (cm)	162.5 ± 10.8	154.1 ± 6.9 **	170.8 ± 6.4
Body mass (kg)	50.2 ± 12.7	41.4 ± 6.6 **	58.9 ± 11.2
30m Sprint max (seconds)	4.8 ± 0.3	5.0 ± 0.2 **	4.6 ± 0.3
Age to/from PHV (years)	-0.2 ± 1.2	-1.2 ± 0.5 **	0.9 ± 0.6

Note: *Significantly different from more mature group (p<0.05).

**Significantly different from more mature group (p<0.01).

Table 1.2. Perceived Readiness (PR) recovery scale used to guide inter-interval recovery periods.

7- Exhausted	(unable to exercise)
6- Very tired	(unable to exercise at the required intensity)
5- Tired	(not yet able to exercise at the required intensity)
4- Adequately recovered	(able to exercise at the required intensity)
3- Well recovered	(able to exercise above the required intensity)
2- Very well recovered	(well able to exercise above the required intensity)
1- Fully recovered	(able to exercise at maximal intensity)

Table 1.3. Mean values for all participants during repeated sprint trials with standardised recovery (SR), self-guided recovery facilitated by the use of a perceived readiness scale (PR), and self-guided recovery with no external cue (NEC).

Variable	SR n=28	NEC n=28	PR n=28
Sprint decrement (%)	2.7 ± 1.7	5.1 ± 3.7 **	3.7 ± 2.1 *
Sprint mean (seconds)	4.9 ± 0.2	5.0 ± 0.3 *	5.0 ± 0.3 *
Recovery average (seconds)	30.0 ± 0.0	19.7 ± 7.1 **	21.2 ± 6.6 **
Recovery CV (%)		21.1 ± 8.7	22.9 ± 7.7
Mean HR (bpm)	165.6 ± 10.6	172.2 ± 9.0 **	171.8 ± 8.2 *

Note: *Significantly different from SR trial (p<0.05).

**Significantly different from SR trial (p<0.01).

Table 1.4. Mean values for the more and less mature group during repeated sprint trials with standardised recovery (SR), self-guided recovery facilitate by the use of a perceived readiness (PR) scale and self-guided recovery with no external cue (NEC).

Variable	More mature group (n=14)			Less mature group (n=14)		
	SR	NEC	PR	SR	NEC	PR
Sprint decrement (%)	3.2 ± 2.1	4.4 ± 3.8	3.9 ± 2.3	2.2 ± 1.4	5.8 ± 3.6 *	3.6 ± 1.9 *
Sprint average (seconds)	4.7 ± 0.2 $\pi\pi$	4.9 ± 0.2 $\pi\pi$ *	4.8 ± 0.2 $\pi\pi$	5.1 ± 0.2	5.2 ± 2 *	5.2 ± 0.2 *
Recovery duration (seconds)	30.00 ± 0.0	21.6 ± 6.5 **	22.8 ± 4.8 **	30.0 ± 0.0	17.8 ± 7.4 **	19.5 ± 7.8 **
Recovery CV (%)		19.4 ± 5.1	21.9 ± 8.1		22.8 ± 11.2	23.9 ± 7.4
Heart rate (bpm)	167.2 ± 9.0	172.2 ± 8.5 *	172.3 ± 8.3 *	164.1 ± 10.6	172.2 ± 8.5 **	171.4 ± 8.4 *

Note: * Significantly different from SR (p<0.05)

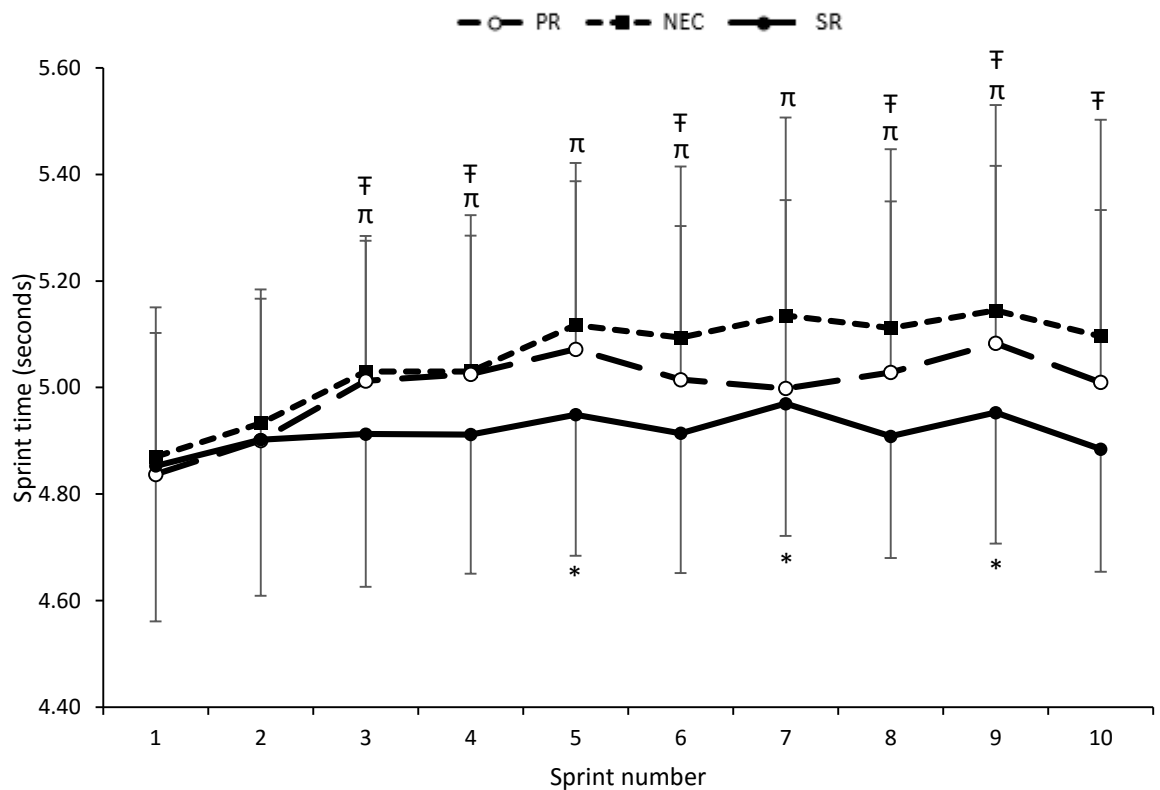
** Significantly different from SR (p<0.01)

$\pi\pi$ Significant between group difference (p<0.01)

Figures

Figure 1.1. Sprint times during repeated sprint trials with standardised recovery (SR), self-guided recovery facilitated by the use of a perceived readiness (PR) scale, and self-guided recovery with no external cue (NEC) for the both groups combined (Figure 1a), the more mature group (Figure 1b) and the less mature group (Figure 1c).

a)

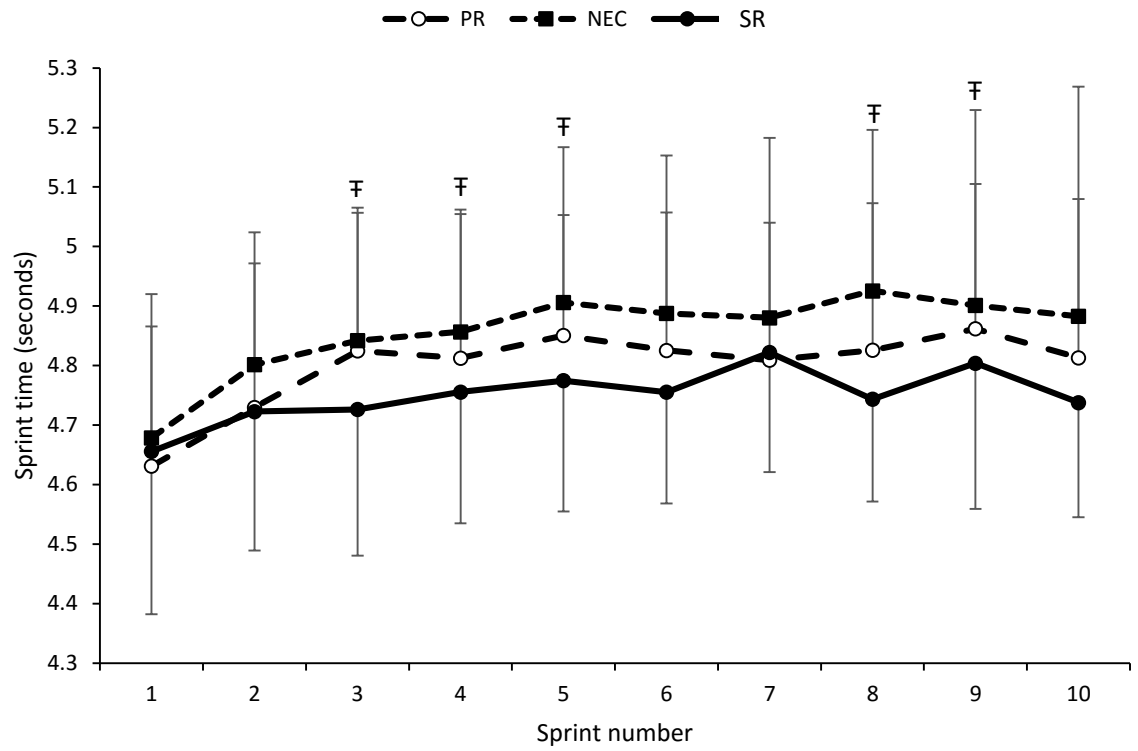


Note: * Significantly different from sprint 1 in SR trial ($p < 0.05$)

† Significantly different from sprint 1 in PR trial ($p < 0.05$)

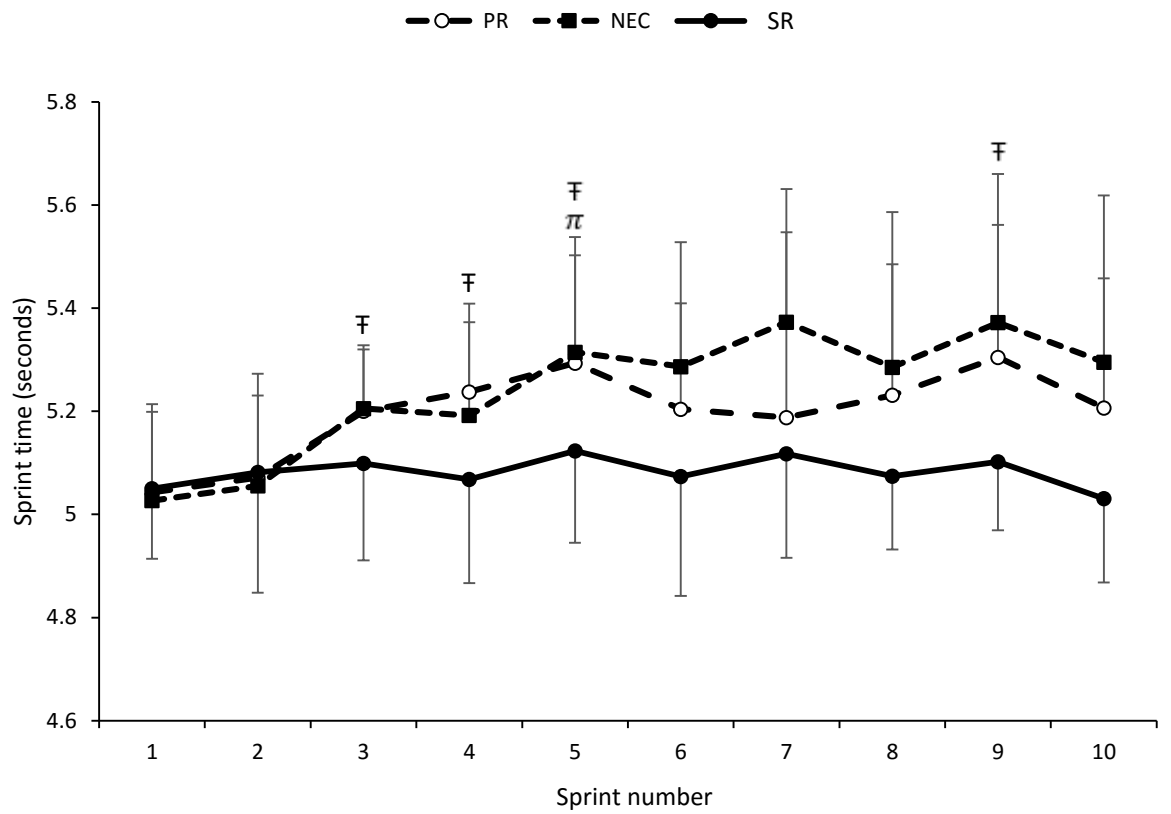
π Significantly different from sprint 1 in NEC trial ($p < 0.05$)

b)



Note: F Significantly different from sprint 1 in PR trial ($p < 0.05$)

c)

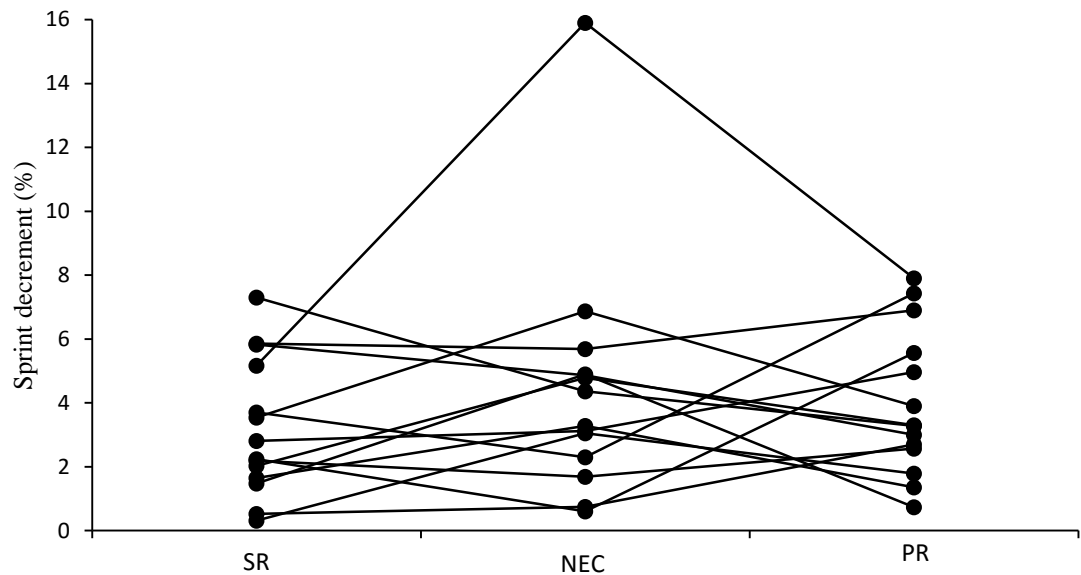


Note: F Significantly different from sprint 1 in PR trial ($p < 0.05$)

π Significantly different from sprint 1 in NEC trial ($P < 0.05$)

Figure 1.2. Percentage sprint decrement during repeated sprint exercise with standardised recovery periods (SR), self-guided recovery periods facilitated by the use of a perceived readiness scale (PR), and self-guided recovery with no external cue (NEC) for individual participants in the more mature group (a) and less mature group (b).

a)



b)

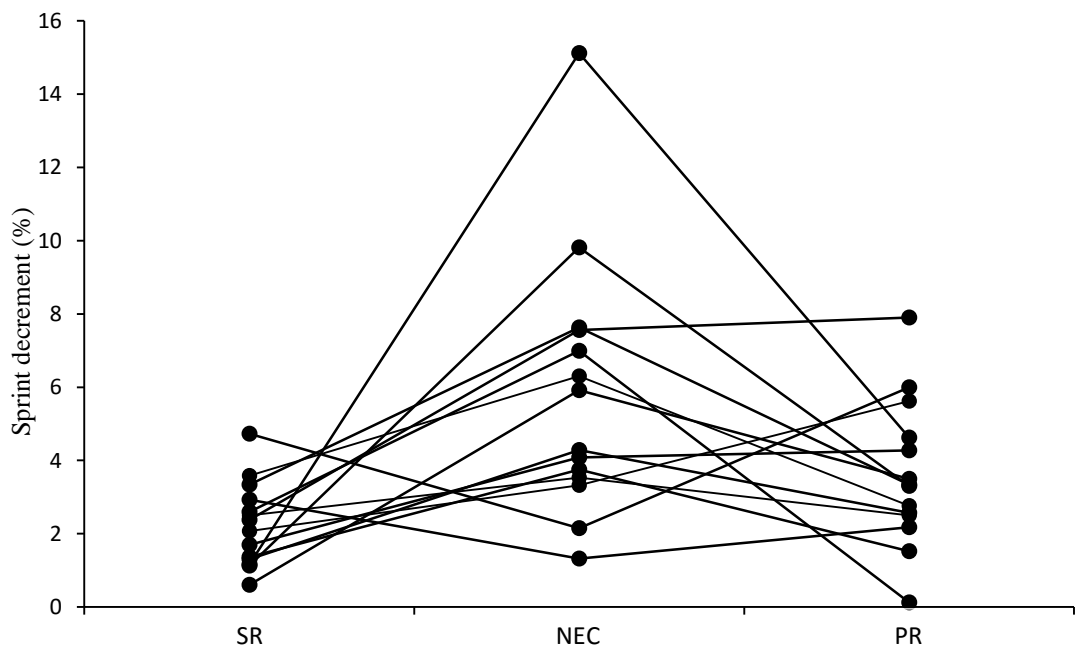


Figure 1.3. Inter-interval recovery durations during repeated sprint trials with self-guided recovery facilitated by the use of a perceived readiness scale (PR), and with no external cue (NEC) for both groups combined.

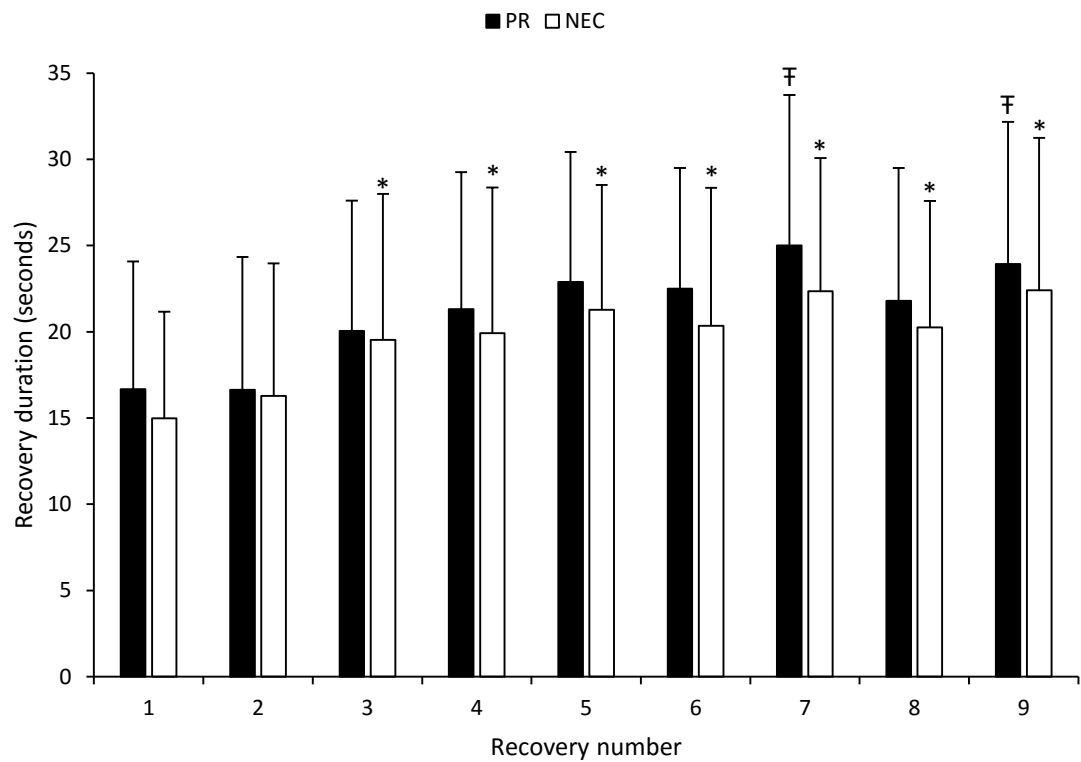
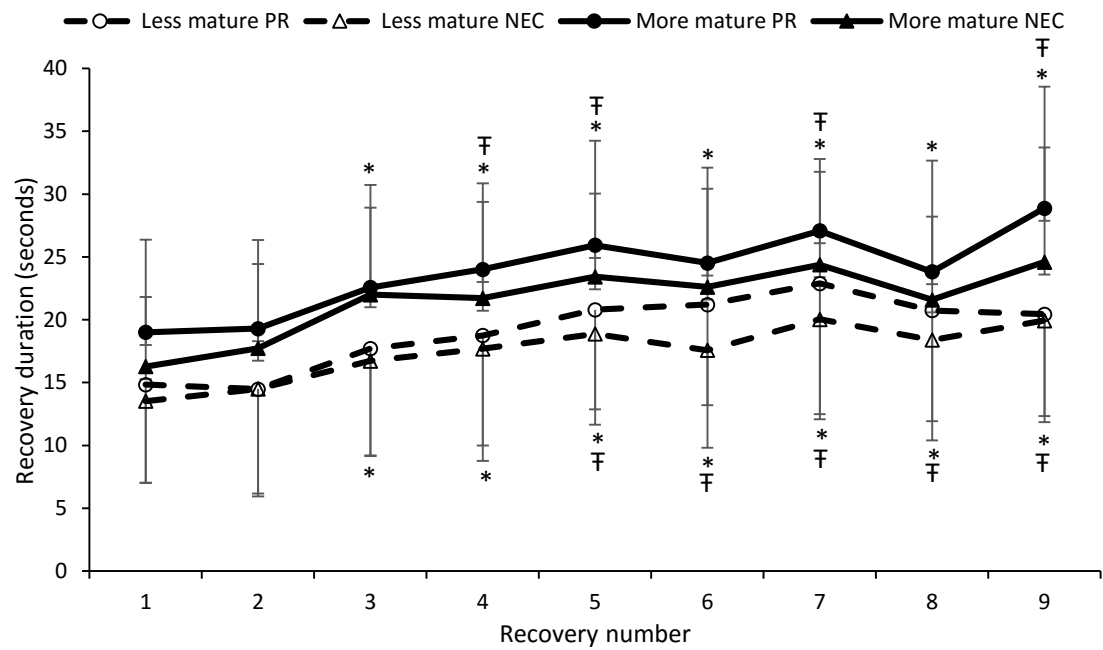


Figure 1.4. Inter-interval recovery durations during intermittent sprint exercise with self-guided recovery facilitated by the use of a perceived readiness scale (PR), and with no external cue (NEC) in the less and more mature group.



Note: F Significantly different from sprint 1 in PR trial (p<0.05)

* Significantly different from sprint 1 in NEC trial (P<0.05)

Chapter 4 – Study Two – An Investigation into the Physiological, Neuromuscular and Perceptual Responses to a Self-regulated Repeated Sprint Task in Elite Youth Association Football Players

4.1 - Introduction

Over the last decade, the topic of repeated sprint training has received increased attention within the scientific literature. A bout of repeated sprint exercise (RSE), which is characterised by short duration sprints (≤ 6 seconds) interspersed by short recovery periods (≤ 60 seconds) (Buchheit *et al*, 2013), induces a high degree of physiological, neuromuscular, metabolic and perceptual stress (Spencer *et al*, 2005). Performing repeated sprint exercise (RSE) has been shown to induce numerous physiological and biomechanical adaptations relevant to team sport athletes (Taylor *et al*, 2015), including improvements in maximum sprint speed (Buchheit *et al*, 2008), aerobic capacity (Bishop *et al*, 2011) and jump height (Buchheit *et al*, 2008). As a result, repeated sprint training is regularly implemented by practitioners working with team sport athletes as a time-efficient means of improving these physical attributes.

The traditional approach to repeated sprint training is to separate sprints using standardised recovery periods, usually of between 20-30 seconds in duration (Bravo *et al*, 2008; Castagna *et al*, 2008; Buchheit *et al*, 2010). However, using such an approach may not account for the individual differences in the capacity to recover between sprints in order to reproduce maximal sprint performance, and as a result, desired training outcomes may be compromised if inadequate or excessive recovery periods are given (Edwards *et al*, 2011). In light of this, recent studies have employed a self-paced, individualised approach to RSE, whereby participants are given the freedom to self-regulate recovery periods based on their perceived readiness for recommencing sprint exercise (Glaister *et al*, 2010; Phillips *et al*, 2014). Using this approach, participants are required to interpret physiological feedback in order to determine their readiness for recommencing subsequent sprints whilst using the minimal amount of recovery time required to maintain performance (Phillips *et al*, 2014). Edwards *et al* (2011) suggested that using self-regulated recovery periods may better account for the physiological, psychological and environmental influences that may contribute to the variability in inter-interval recovery rates between athletes (Edwards *et al*, 2011). Furthermore, allowing team sport athletes to self-regulate recovery during RSE may more closely replicate game demands more as

players are required to respond to the physiological demands within the confines of successfully completing the task in accordance with their capabilities, an aspect that has been suggested as important during team sports such as association football (Edwards & Noakes, 2009).

However, while these studies have suggested that RSE with self-guided recovery may be a useful alternative to repeated sprint training in adults (Glaister *et al*, 2010), no studies have investigated this form of training in young athletes. Research in this area may give insight into the applicability of self-regulated RSE in younger age groups. Furthermore, integral to the implementation of training interventions which aim to induce specific adaptations is an understanding of the isolated acute physiological, metabolic, neuromuscular and perceptual responses evoked by specific training modalities. Despite this, limited data exists exploring these responses to repeated sprint exercise (RSE) with self-guided recovery periods. Information on these responses may assist practitioners in making more informed decision making when considering implementing this form of training in young athletes with the aims of inducing specific training outcomes.

Thus, the aim of the present study was to assess the physiological, metabolic, perceptual, neuromuscular and performance responses of a repeated sprint task with self-guided recovery compared with standardised recovery intervals in elite youth association football players.

4.2 - Methods

Participants

Eleven male elite youth football players from a professional Scottish football academy were recruited to the study. Physical characteristics of the players are presented in Table 2.1. All players undertook 10-15 hours of football-specific training, strength and conditioning, and competitive match play per week. Participants and, where appropriate their guardian(s), provided written informed consent prior to the data collection procedure. Participants agreed to abstain from performing strenuous exercise in the 24 hours prior to data collection. The study received institutional ethics approval and conformed to the declaration of Helsinki.

Experimental design

Baseline testing took place for maximum 30m sprint speed, body mass, and stature (Table 2.1). In order to reduce the impact of any learning effect, participants were habituated with the study protocol, including countermovement jump (CMJ) technique, and the repeated sprint protocol with both self-guided and externally controlled passive recovery prior to the data collection.

Before the repeated sprint trials, participants completed a standardised ten minute warm-up consisting of running and dynamic mobility exercises. Pre-trial CMJ measurements were obtained in a laboratory setting using a force plate before participants moved to an outdoor synthetic pitch located within 20m of the laboratory to perform the repeated sprint assessments. All of the trials were performed at the same time of day during mild UK spring months (mean \pm SD: ambient temperature = $14.8^{\circ}\text{C} \pm 2.8^{\circ}\text{C}$, relative humidity = $71\% \pm 6.8\%$, wind flow velocity = $11.4\text{mph} \pm 5.2\text{mph}$).

Each repeated sprint trail consisted of 10 x 30m sprints interspersed with passive recovery. Participants began each sprint 0.5m behind the infrared timing gate (SmartSpeed, Fusion Sport, Australia) from a standing start. Following each sprint, participants were instructed to decelerate and walk back to the starting line before performing the subsequent sprint. The repeated sprints were performed under the following two conditions:

- 1) A standardised recovery (SR) trial, in which all participants were given 30 seconds of recovery between sprints, as used in the Castagna *et al* (2008) study.

- 2) A self-guided recovery (SG) trial, in which participants were allowed to determine the recovery duration between each sprint. During this trial, participants were instructed to use the minimal amount of recovery time they felt was required in order to achieve their fastest 30m sprint time during successive sprints, and were given no external cue on when to recommence sprint exercise.

Participants were unaware as to which experimental trial they were to perform until after the warm-up. Following the repeated sprint protocol, players returned to the laboratory for the assessment of physiological and neuromuscular parameters. Physiological and neuromuscular measurements were taken at two, five and seven minutes following the repeated sprint assessments, with physiological measurements taken prior to neuromuscular at each time point. Perceptual responses (overall session RPE) were measured two minutes following the assessments. At least one week was given between each sprint trial.

Outcome measures

Repeated sprint performance. Repeated sprint performance was measured through percentage sprint decrement ($100 \times (\text{total sprint time} \div \text{ideal sprint time}) - 100$) (Glaister *et al*, 2008) and average sprint time. Sprint times during both trials and recovery times during the SG trial were recorded using infrared timing gates (SmartSpeed, Fusion Sport, Australia).

Neuromuscular measurements. Neuromuscular responses were measured through CMJ flight-to-contraction time (i.e. the time spent during the total eccentric, isometric and concentric phase of the jump in relation to the time spent off the ground) and peak force, with variables measured using a force platform (MuscleLab Force Platform, Ergotest Innovation, Norway). During each CMJ, participants were instructed to keep their hands held in place on the hips, and to squat until the femur was parallel with the floor immediately prior to the concentric phase of the jump. Any jump that did not conform to the correct technique was discarded from the analysis and repeated. Measurements were taken before and two, five and seven minutes following the trials.

Physiological measurements. Mean HR ($\text{b} \cdot \text{min}^{-1}$), peak HR, and HR recovery were recorded using a polar HR monitor (H7 HR monitor, Polar Electro, Finland). HR recovery was calculated as the HR at the end of each sprint minus HR at the end of each recovery period to the nearest second, with values analysed visually using an excel spreadsheet. The average of these values was used across the nine recovery periods.

Blood lactate measurements were obtained from the fingertips of the right hand while players were in a seated position, and were taken at two, five and seven minutes following the repeated sprint trials, before being assessed using a lactate analyser (Biosen C-line glucose and lactate analyser, EKF-Diagnostics, Germany).

Perceptual measurements. As an index of the overall subjective perceptions of exertion during the sprints, RPE was assessed with the Borg 15 point scale (Borg, 1970), with measurements being taken 2 minutes following the repeated sprint trial. Subjects were habituated with the use of the scale prior to data collection.

Statistical analysis

The number of participants used for the study was determined based on the results from the study presented in chapter 3. In this study, using self-selected recovery periods with no external feedback resulted in a significant increase in percentage sprint decrement compared with using standardised recovery periods ($ES = .82$). Using these values, a power calculation (G*power, version 3.0, Germany) revealed that 11 participants would be required to achieve statistical significance with $\alpha = 0.05$ and 80% power. Paired student t-tests were used to compare differences between trials in performance, physiological and neuromuscular measures. Wilcoxon signed-rank test was used to assess between trial differences in RPE. A repeated measures ANOVA with Bonferonni corrections was used to assess within-trial changes in blood lactate, CMJ F:C time and peak force, as well as to analyse within-trial changes in sprint time. In the event of a significant main effect, Bonferonni *post-hoc* analyses was to assess differences. Cohens d effect sizes (ES) were used to quantify the magnitude of mean differences between trials and between blood lactate and CMJ measurement intervals (*trivial* = $d \leq 0.1$; *small* = $d > 0.1, < 0.5$; *moderate* = $d > 0.5, < 0.8$; *large* = $d \geq 0.8$) (Cohen, 1992). Statistical significance was accepted at $p < 0.05$. All results are displayed as mean \pm SD unless otherwise stated.

4.3 - Results

Sprint performance

Performance measures during the SR and SG trial are displayed in Table 2.2. Mean sprint time was significantly longer during the SG trial in comparison with the SR trial ($p < 0.05$; $ES = .4$). No significant difference was found between percentage sprint decrement in the SR and SG trial ($ES = .51$) (Table 2.2). The average recovery duration taken during the SG trial was significantly less than during the SR trial ($p < 0.01$; $ES = 2.6$) (Table 2.2).

Average sprint times during each of the 10 sprints in the SR and SG trial are displayed in Figure 2.1. No significant within-trial differences were found in sprint time throughout the ten sprints in either trial.

Physiological, neuromuscular and perceptual responses

Average heart rate responses to the SR and SG trials are provided in Figure 2.2. No significant difference was found between the SR and SG trials for average HR (167.2 ± 8.4 bpm vs 172.8 ± 9.8 bpm) ($ES = .52$) or peak HR (180.0 ± 12.17 bpm vs 183.1 ± 13.2 bpm) ($ES = .24$; Figure 2.3). Although not statistically significant, there was a general trend towards HR recovering to a greater extent in the SR trial compared with the SG trial ($p = .055$) (Figure 2.4; $ES = .96$).

Figure 2.5 displays the blood lactate concentrations post-exercise. Blood lactate measured two minutes following RSE tended to be higher in the SG trial (8.8 ± 2.6 mmol/l vs 7.0 ± 2.2 mmol/l; $p = 0.059$; $ES = .75$). Blood lactate was significant higher in the SG trial at five minutes ($p < 0.05$; $ES = .59$) and seven minutes ($p < 0.05$; $ES = .62$) compared with the SR trial.

No significant main effect for time on blood lactate was found in the SR trial ($F_{1,11} = 1.823$; $p > 0.05$) (2-5 minutes $ES = .50$; 2-7 minutes $ES = .51$; 5-7 minutes $ES = .06$). In contrast, a significant main effect for time on blood lactate was found in the SG trial ($F_{1,11} = 6.687$; $p < 0.01$), with *post-hoc* comparisons revealing that blood lactate concentration two minutes post exercise was significantly higher than seven minutes post exercise in the SG trial ($p < 0.05$; $ES = .7$), with no difference between two and five, or five and seven minutes (2-5 minute $ES = .68$; 2-7 minute $ES = .69$; 5-7 minute $ES = .02$) (Figure 2.5).

No significant within-trial or between-trial differences were found in CMJ peak force (Figure 2.6) or F:C time (Figure 2.7) at any time point (all $ES \leq .1$). Similarly, no

significant differences were found between post exercise RPE measured following the SR and SG trials (Table 2.2).

4.4 - Discussion

The primary aim of the present study was to compare the physiological, perceptual, neuromuscular and performance responses to a repeated sprint task with self-guided recovery compared with standardised recovery intervals in elite youth football players. The data demonstrate that using self-regulated recovery intervals resulted in a higher physiological and metabolic demand and impaired repeated sprint performance when compared with 30-second standardised recovery intervals.

Performance during RSE is influenced by the duration of the inter-interval recovery periods (Balsom *et al*, 1992). In the present study, participants used significantly less recovery time during the SG trial than was provided during the SR trial (19.7 vs 30.0 seconds, respectively) (Table 2.2). As a result of the shorter recovery periods used, performance was impaired when compared with the SR trial, with a significantly higher average sprint time, and a *moderately* higher percentage sprint decrement during the SG trial (Table 2.2), suggesting that participants employed an insufficient amount of recovery to allow performance to be maintained throughout the sprints. Conversely, when 30 second standardised recovery periods were employed, participants were able to maintain a consistent performance throughout the sprints (Figure 2.1).

Comparison of the blood lactate measurements after SG and SR trials revealed that lactate was *moderately* higher during the SG trial at two, five and seven minutes post-exercise (Figure 2.5). These results are in agreement with the studies of Balsom *et al* (1992) and Wooton & Williams (1983), who similarly observed higher blood lactate concentrations following RSE with shorter recovery intervals compared with longer recovery intervals. The higher blood lactate observed during the SG trial suggests that anaerobic glycolysis may have been stimulated to a greater extent than in the SR trial. The primary fuel during short-duration sprint exercise is phosphocreatine (PCr), which represents the most immediate reserve for ATP rephosphorylation (Girard *et al*, 2011). An important function of the recovery process is therefore to replenish PCr stores towards resting values in order to allow ATP to be resynthesised at the required rate. However, given that the restoration of PCr is a time-dependent process (Balsom *et al*, 1992), it could be postulated that the shorter recovery intervals used during the SG trial resulted in incomplete restoration of PCr stores, and thus placed a greater demand on anaerobic glycolysis to maintain ATP turnover during successive sprints, resulting in higher blood lactate concentrations.

Moreover, analysis of HR data from the present study suggests that using self-guided recovery intervals imposed a higher physiological demand on the players, with average HR *moderately* higher during the SG trial (Figure 2.3), while effect sizes suggested differences of a *large* magnitude between HR recovery during the SG and SR trials (4 bpm vs 9bpm, respectively) (Figure 2.4). The shorter recovery intervals resulted in a progressive increase in HR throughout the SG trial, as displayed in figure 2.2. During RSE, the contribution of aerobic metabolism increases during successive sprints as PCr stores are depleted and the contribution of anaerobic glycolysis decreases (Gaitanos *et al*, 2003). In turn, increased aerobic contribution requires an increased oxygen supply and cardiorespiratory demand (Phillips *et al*, 2014), which may explain the higher HR values observed during the SG trial. Additionally, the higher average HR values displayed during the SG trial may have been the result of an increase in the demand for oxygen in order to metabolise lactate and/or resynthesise PCr (Gatianos, 1993).

Another interesting finding from the study was that despite the higher physiological demand and increased fatigue found during the SG trial, no differences were observed between RPE measured following either trial. This is surprising given the higher HR and blood lactate during the SG trial, both of which have been correlated with RPE (Coutts *et al*, 2009), as well as the higher fatigue found during the SG trial. While it is difficult to speculate on why this may have occurred, this may have been due to the post-session RPE measure being insensitive to the increased demands imposed during the SG trial. Alternatively, the similar RPE may suggest that participants were unaware of the higher physiological stress and fatigue induced during the SG trial compared with the SR trial. During endurance type exercise, it has been suggested that externally paced work presents a higher perceptual demand than self-regulated work of matched intensity (Lander *et al*, 2009). However, given that the duration of recovery intervals was significantly different between the two trials, it is difficult to elucidate whether the same response occurs during repeated sprint exercise with self-guided recovery, and this presents an area for future research.

Despite the heightened fatigue response to the SG trial, no within-trial or between-trial differences were found between CMJ peak force (Figure 2.6) and flight-to-contraction time (Figure 2.7). In contrast, Morcillo *et al* (2015) found that a similar repeated sprint protocol (12 x 30m sprints, 30 seconds recovery) induced significant neuromuscular fatigue measured through post-exercise CMJ height in adults. However, given that adolescents display an enhanced ability to recover more quickly from maximum intensity

exercise compared with adults (Ratel *et al*, 2006), the two minutes given between the end of the repeated sprint trials and the post-test CMJ measure may have been sufficient to allow recovery of neuromuscular function, which may explain the discrepancies between the studies.

The main limitation of the present study was that due to logistical constraints, the repeated sprint trials were conducted on an outdoor synthetic pitch. Thus, environmental influences such as temperature, humidity, wind and precipitation may have influenced performance and the physiological and perceptual responses to the trials. However, all of the trials were performed at the same time a day during mild UK spring months, and the temperature, humidity and wind flow velocity recorded displayed little variation throughout the study. Furthermore, the temperature and humidity values fall below previously identified thresholds known to influence repeated sprint performance (Drust *et al*, 2005), suggesting that any environmental influences on the results were trivial.

In conclusion, the present study has displayed that using self-guided recovery periods during repeated sprint exercise has a significant impact on performance and the physiological and metabolic responses to RSE when compared with a traditional standardised recovery period in young athletes. Specifically, participants employed significantly less recovery time during self-guided recovery trials, but as a result displayed a higher degree of fatigue and physiological and metabolic stress.

Practical applications

During repeated sprint training, variables such as sprint length, number of sprints and duration of recovery period can be manipulated in order to achieve a number of desired outcomes (Bishop *et al*, 2011). For example, it has been suggested that when improvements in maximum running speed are sought, using longer recovery durations may be more effective in allowing athletes to maintain peak sprint velocity (Taylor *et al*, 2015). Alternatively, using shorter recovery periods has been recommended when the aim is provide a high physiological stress in order to induce improvements in aerobic and anaerobic function (Taylor *et al*, 2015). Given the shorter recovery intervals and higher physiological and metabolic demand imposed on the participants during the self-guided trial in the current study, it could be suggested that this mode of repeated sprint training may be effective when improvements in aerobic and anaerobic function are sought. In contrast, standardised recovery periods may be a more effective tool when the aim of the

session is maintain peak sprint velocity. In order to further investigate this mode of RSE, future research should assess the chronic responses to repeated sprint training with self-guided and standardised recovery periods through physiological and/or field based measures.

Tables

Table 2.1. Physical characteristics of participants.

Variables	Initial characteristic n=11
Age (years)	13 ± 0.9
Height (cm)	167.9 ± 9.8
Body mass (kg)	56.1 ± 15.8
30m sprint max (seconds)	4.8 ± 0.3

Table 2.2. Performance, recovery and perceptual variables during a repeated sprint task with standardised recovery (SR) periods or a self-guided (SG) recovery periods.

Variable	SR n=11	SG n=11	Effect sizes
Sprint decrement (%)	3.4 ± 1.3	4.3 ± 2.1	.51
Sprint average (seconds)	4.9 ± 0.3	5.0 ± 0.3 *	.4
Recovery average (seconds)	30.0 ± 0.0	19.7 ± 5.6 **	2.6
RPE	12.8 ± 1.4	12.8 ± 1.7	0.0

Note: *Significantly different from SR trial ($p < 0.05$).

**Significantly different from SR trial ($p < 0.01$).

Figures

Figure 2.1. Average sprint times during a repeated sprint task with standardised recovery (SR) periods and self-guided (SG) recovery periods.

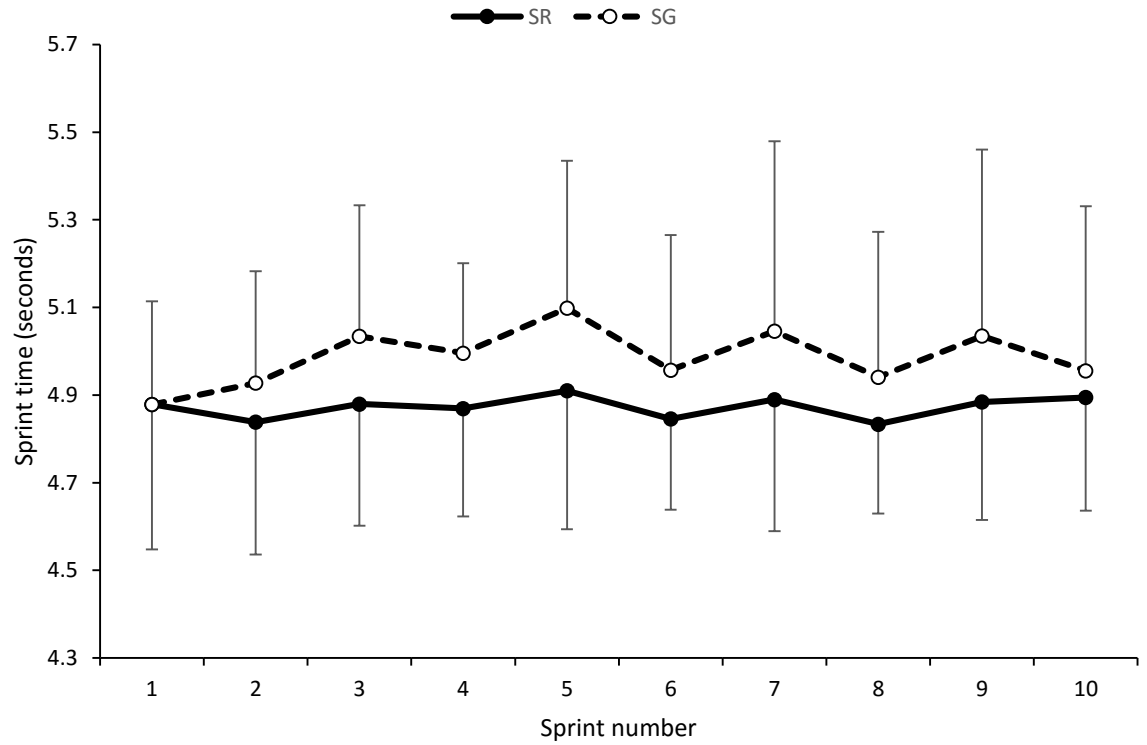


Figure 2.2. Average heart rate responses during a repeated sprint task with standardised recovery (SR) periods and self-guided recovery periods (SG) from a representative participants.

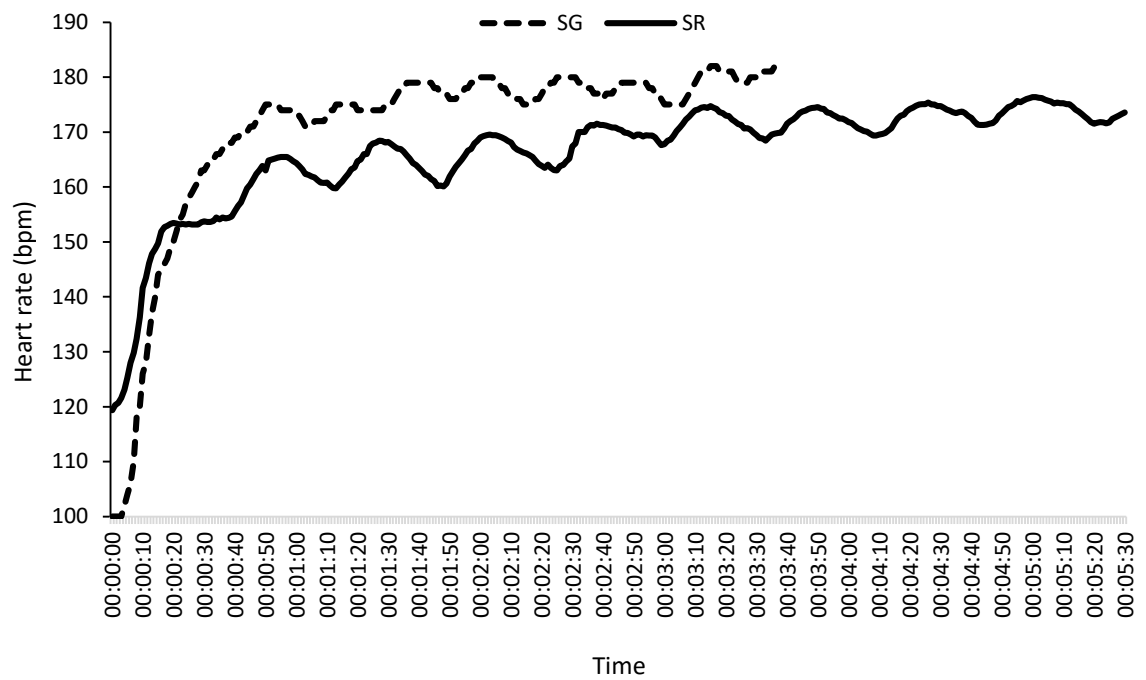


Figure 2.3. Average heart rate and peak heart during a repeated sprint task with standardised recovery periods (SR) and self-guided recovery periods (SG).

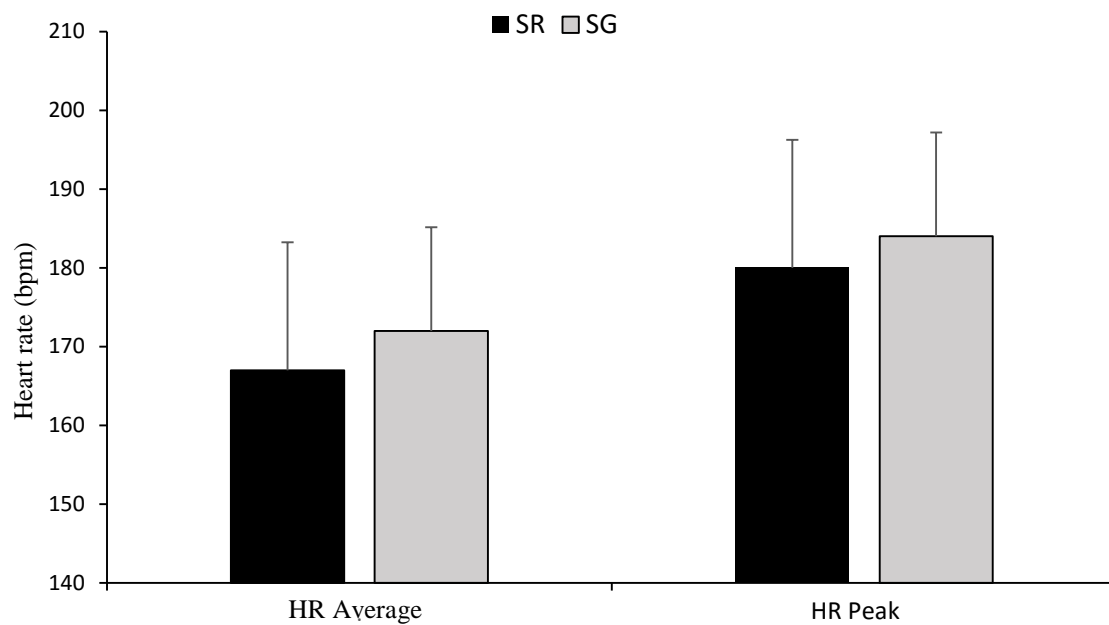


Figure 2.4. Average heart rate recovery between sprints during a repeated sprint task with standardised recovery periods (SR) and self-guided recovery periods (SG).

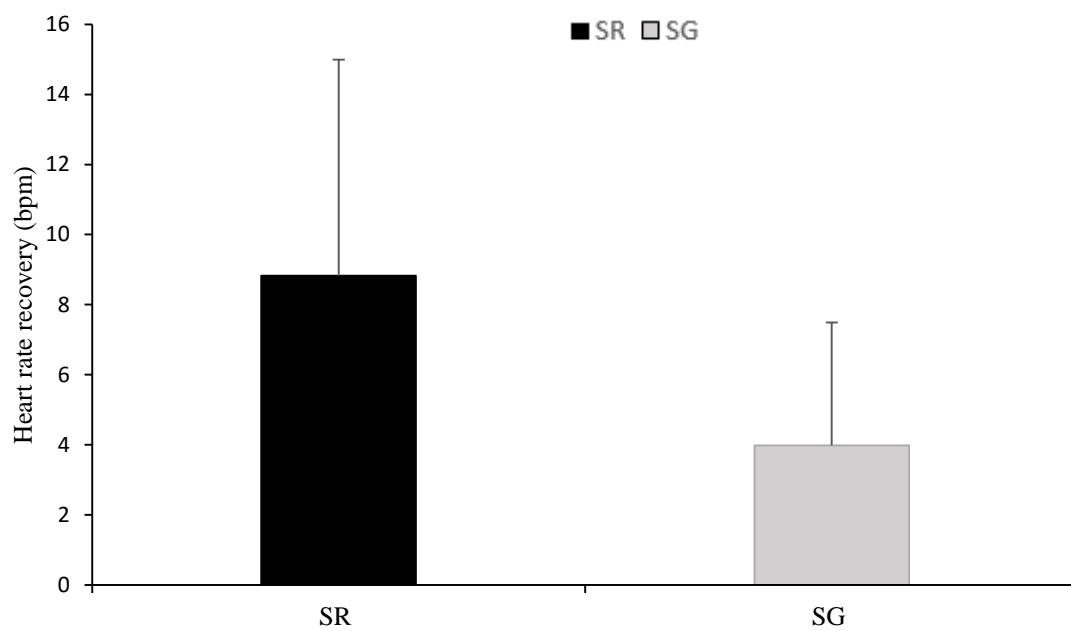
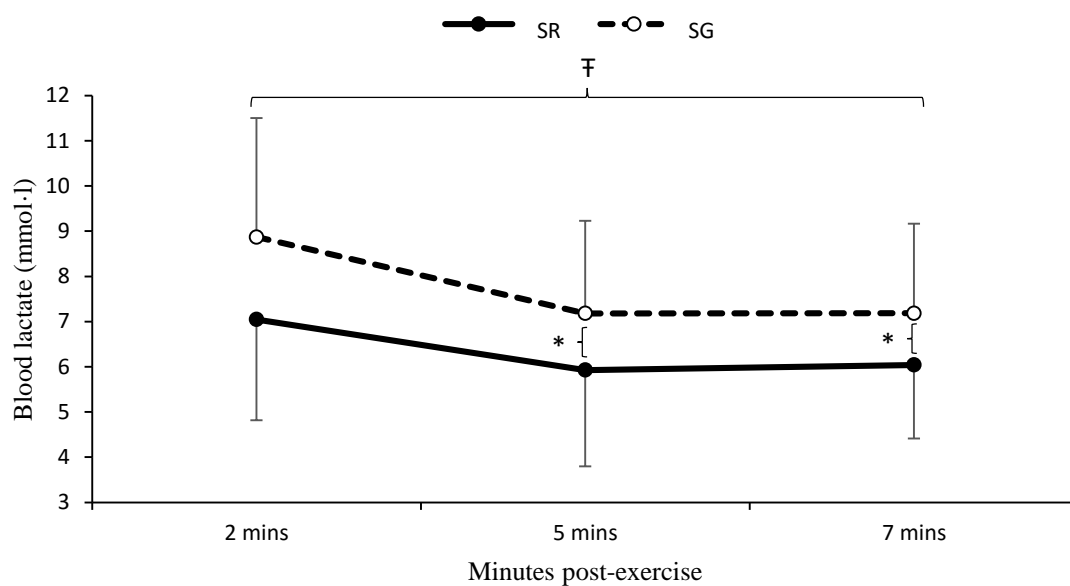


Figure 2.5. Blood lactate concentration 2, 5 and 7 minutes following a repeated sprint task with standardised recovery periods (SR) and self-guided recovery periods (SG).



Note: *Significant between trial difference ($p < 0.05$)

‡ Significant within trial difference during SG trial ($p < 0.05$)

Figure 2.6. Countermovement jump (CMJ) peak force before and 2, 5 and 7 minutes following a repeated sprint task with standardised recovery (SR) periods and self-guided (SG) recovery periods.

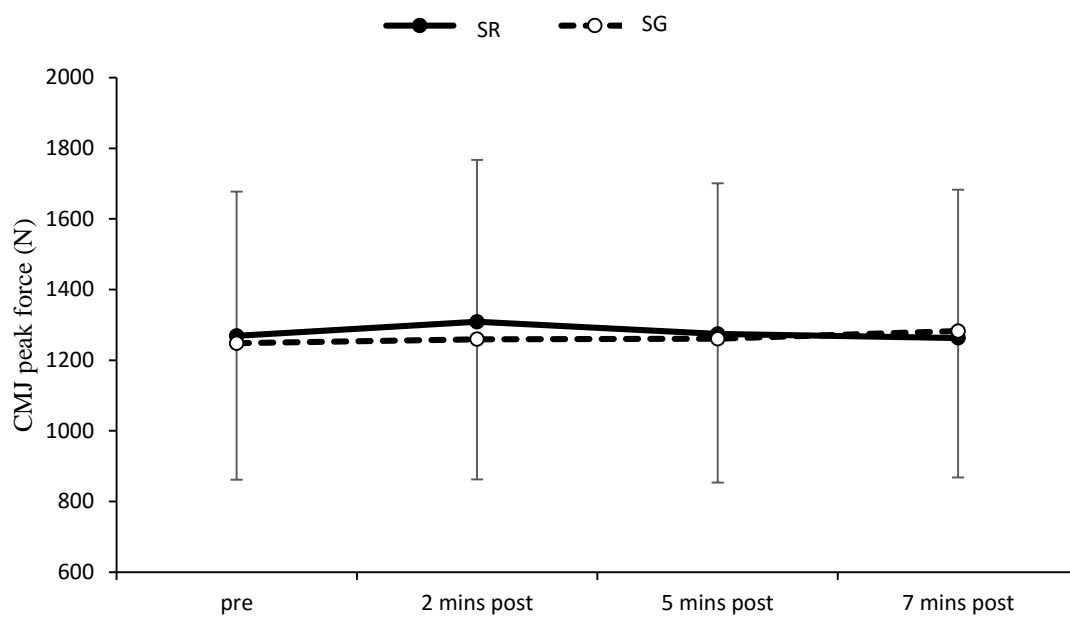
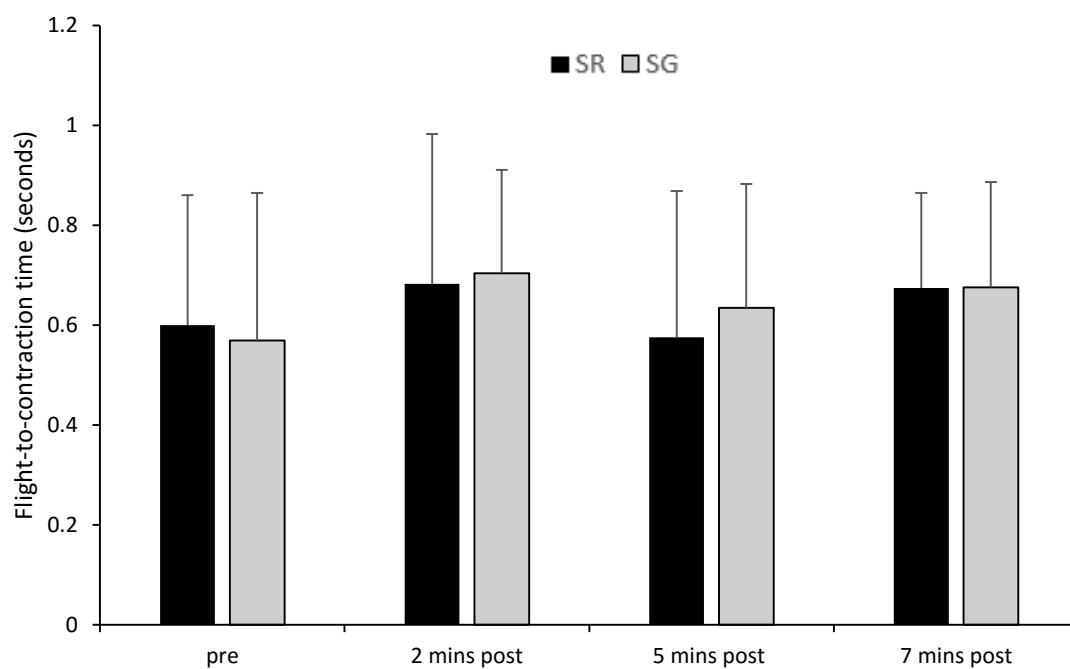


Figure 2.7. Countermovement jump (CMJ) flight-to-contraction time before and 2, 5 and 7 minutes following a repeated sprint task interspersed by a standardised recovery (SR) period and a self-guided (SG) recovery periods.



Chapter 5 – Conclusions

The primary aim of the present thesis was to examine the influence of using a novel, individualised approach to RSE - whereby participants are given the freedom to self-regulate their own recovery periods - on performance and physiological responses in elite youth footballers. The results of the study presented in Chapter 3 displayed that using self-regulated recovery periods has a significant impact on performance when compared with a 30-second standardised recovery period. When participants were asked to self-determine their recovery intervals with no external feedback, they used a significantly shorter recovery period than was given during standardised recovery trial. Despite having autonomy over recovery periods, the participants were unable to maintain sprint performance by effectively manipulating recovery duration. As a result, average sprint duration was significantly longer, and percentage sprint decrement significantly higher than during the standardised recovery trial. This was likely the result of participants underestimating the recovery time required to maintain sprint performance.

The results from the study in Chapter 3 are in contrast to those from studies conducted in adults (Glaister *et al*, 2010; Phillips *et al*, 2014). These studies found that participants were able to maintain a consistent sprint performance when using self-determined recovery periods. However, the recovery intervals used were longer than would typically be allowed during repeated sprint protocols with the same number of sprint repetitions and distances (Glaister *et al*, 2008), whereas participants in the study in Chapter 3 used significantly shorter recovery periods than was provided during the standardised recovery trial. While previous research has displayed that children have a higher propensity for recovery between repeated sprints when compared with adults (Ratel *et al*, 2002) due to a number of physiological mechanisms, the ability to self-regulate recovery is also influenced by cognitive processes, such as planning, anticipation, temporal awareness and logical reasoning. Thus, the lower level of cognitive development in participants in the current study compared with adult populations may explain the contrasting results. As cognitive development was not measured in the study presented in Chapter 3, further work is required to understand how this variable might affect performance in tasks requiring the regulation of recovery duration.

The study in Chapter 3 also examined the influence of using a perceived readiness scale to assist participants in self-guiding recovery intervals. No significant differences were found between the average recovery duration or performance measures in the self-guided

recovery trials with or without the use of the perceived readiness scale. While Edwards et al (2011) proposed that using scalar methodologies may be an effective tool to guide inter-interval recovery durations during high-intensity interval training in adult populations, the results from Chapter 3 suggest that using such methodologies may be less effective in young athletes.

A secondary aim of the study in Chapter 3 was to assess the influence of maturation performance during the repeated sprint trials. Despite exhibiting less fatigue than the more mature group during the SR trial, less mature players displayed impaired performance during repeated sprints when self-guided recovery was used with NEC compared with the more mature group. During this trial, the less mature players used a consistently shorter recovery period, suggesting that while less mature players may be physiologically better equipped to resist fatigue during RSE, they display an impaired ability to accurately interpret physiological feedback within the context of the task. Given that the ability to determine recovery intervals in the context of the task is likely to be influenced by cognitive factors, it may be that the reduced level of intellectual development and/or experience may have contributed to the impaired performance during the NEC trial in the less mature group.

Chapter 4 of the thesis provides insight into the physiological, neuromuscular and perceptual responses to RSE with self-guided recovery intervals compared with a 30-second standardised recovery period. The results of the study suggest that using self-guided recovery intervals results in an elevated fatigue response, with longer average sprint times and higher percentage sprint decrement compared with the SR trial. Furthermore, using self-guided recovery imposed a higher physiological and metabolic stress on the participants, with higher average HR and post-exercise blood lactate during the SG trial. This may have been the result of the participants using less recovery time during the SG trial, which may have been insufficient to allow full restoration of PCr stores, thus placing a greater demand on anaerobic glycolysis and aerobic metabolism and resulting in compromised performance.

While the studies presented in the thesis provide novel insight into self-regulated repeated sprint exercise in young athletes, one of the main limitations in both studies was that participants were given only one session to habituate themselves with self-regulated recovery between sprints. Therefore, it is possible that with more practice, participants may have improved their ability to accurately self-guide recovery intervals in order to maintain sprint performance, and this presents an area for future research. Another

limitation to the present thesis was that in Chapter 4, repeated sprint exercise took place on an outdoor synthetic pitch. Therefore, it is possible that environmental conditions may have influenced performance and/or the physiological responses to repeated sprint exercise. However, environmental conditions were monitored throughout and showed relatively little variation during each trial. Thus, any influence on performance were most likely trivial.

Practical Applications and Future Research

From a practical perspective, the studies in the present thesis demonstrate that elite youth association football players have a tendency to underestimate the amount of recovery time required in order to maintain performance during a self-regulated repeated sprint task. As a result, using self-selected recovery periods appears to induce a higher physiological and metabolic stress compared with using a 30-second standardised recovery period. Thus, if the aim of a repeated sprint training session is to impose a high physiological and metabolic stress in order to induce adaptation, using self-selected recovery periods may be a more suitable approach.

Using 30-second standardised recovery periods, participants in the study displayed a better ability to limit fatigue and more closely reproduce maximal sprint performance. Therefore, when maintenance of work is desired, implementing standardised recovery periods, rather than self-selected recovery periods, may be a more suitable approach. However, given that participants in the study were only given one habituation session, and that previous research has displayed that the ability to accurately self-determine recovery periods improves with repeated trials (Glaister *et al*, 2010), it may be that with additional practice participants would be able to more accurately and consistently govern recovery periods. Thus, an interesting area for future research may be to assess the effect of a period of repeated sprint training using self-selected and standardised recovery periods on physiological and/or field-based fitness measures, and on the ability to accurately self-regulate recovery during RSE.

Another approach which might improve the athletes' ability to accurately self-govern recovery periods would be to give feedback on performance following each sprint. Using this approach may allow for players to more precisely determine whether they were underestimating or overestimating the amount of recovery time they required, and in turn improve awareness of their physical capabilities. This represents an area for future research investigations

Chapter 6 – References

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